AUTOPSY STUDY OF RENAL LESIONS IN SNAKE BITE CASES

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Branch – XIV

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DEPARTMENT OF FORENSIC MEDICINE,

MADURAI MEDICAL COLLEGE,

MADURAI – 625 020.

CERTIFICATE

This is to certify that the dissertation entitled **"AUTOPSY STUDY OF RENAL LESIONS IN SNAKE BITE CASES"** is the bonafide work of **Dr.VIJAY BALAJI R** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D., (Forensic Medicine) Branch– 14 examination to be held in May 2019.

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ENDORSEMENT BY THE DEAN

This is to certify that the dissertation entitled **"AUTOPSY STUDY OF RENAL LESIONS IN SNAKE BITE CASES**" is a bonafide and genuine research work done by **Dr. VIJAY BALAJI R,** in partial fulfilment of the requirement for the degree in **M.D., (Forensic Medicine) Branch-XIV** under guidance of **Prof & HOD Dr.T. Selvaraj, M.D.,** Department of Forensic Medicine, Madurai Medical College, Madurai.

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DECLARATION

I, **Dr.VIJAY BALAJI R**, hereby declare that, I carried out this work on **"AUTOPSY STUDY OF RENAL LESIONS IN SNAKE BITE CASES"** at the Department of Forensic Medicine, Government Rajaji Hospital, Madurai, under the guidance of **Prof. Dr. T. SELVARAJ, M.D. D.C.H.,** Head of the Department of Forensic Medicine, during the period of one year from April 2017 to March 2018. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other University or Board either in India or Abroad.

This is submitted to the Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in Forensic Medicine (Branch – 14) to be held in May- 2019.

Dr. VIJAY BALAJI R

Place : Madurai

Date :

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CONTENTS

1. INTRODUCTION

Snakes are elongate, carnivorous, legless reptiles of the suborder ophidia (class - Reptiles, subclass - Lepidosauria, order - Squamata, suborder - ophidia). There are about 3500 species of snake in the world but only about 350 species are venomous and only a minority of these are likely to cause significant envenoming in humans. In India 330 snake species exists, of which 70 are venomous.

Venomous snakes:

All venomous snakes of the world are grouped into five families.

a) Colubridae:

- Fixed and rear fanged (opisthoglyphous)
- Only few are dangerous to humans (Eg: Boomslang, Mountain tracer, Tree snake)
- The family includes about 2/3rd of all snake species on earth
- Includes some non poisonous snake also (Eg: Rat snake)
- About 2/3rd of colubridae have developed a gland called the Duvernoy' s gland

Duvernoy' s gland:

- Found only in colubridae

- Gland is positioned posterior to the eye, encased in a thin cover of connective tissue, and consists mostly of serous cells. A single short duct extends anteromedially from the lumen of the gland to the base of posterior fangs.
- The venom simply flows along a posterior groove into the oral cavity. It is a homologue of venom glands in Viper' s and elapids, yet it is anatomically and functionally distinct.

Differences from a true venom gland:

- Lacks muscles - Duvernoy' s gland is not typically associated with muscles that result in pressurised venom expulsion.
- Histology - Its secretions are toxic, but histologically it is different from viperid and elapid snakes.
- Functions - Secretions of Duvernoy' s gland are not meant to kill prey. Its function are lubrication, digestion, inhibition of oral bacterial growth, neutralisation of microbes.

b) Elapidae:

- Fixed and front fanged (proteroglyphous)
- Cobras, Kraits, Mambas, Coral snakes
- Major global cause of snake bite

c) Atractaspididae:

- Side fanged
- Not a major cause of snake bite

d) Viperidae:

- Folding and front fanged. Fangs are like hollow syringe needles (Solenoglyphous)
- Divided into 4 subfamilies

1. Viperinae (True / Typical /' old world' vipers)

- African puff adders
- European adders
- Gaboon vipers
- Russell' s viper
- Saw scaled viper

2. Crotalinae (Pit viper)

- Eg: Copperheads, cotton mouths, Green pit viper, Malayan pit viper, Rattle snakes.

3. Azemiopinae (Fea' s viper)

4. Causinae (Night adders)

- Most US venomous snakes belong to Crotaline. Both major Indian Vipers belong to Viperinae.

e) Hydrophidae:

- Fixed and front fanged, but habitat is water
- Adapted to aquatic environment morphologically and physiologically
- Possess specialised flattened tails for swimming, valves over both nostrils.
- 2 subfamilies: 1. Lticaudinae (Sea Kraits), 2. Hydrophinae (True sea snakes)
- Mostly seen in warm water of India and Pacific oceans.
- WHO classification:

Category 1: Cause death or serious disability

Category 2: Cause serious effects

Category 3: Serious effects are uncommon

- Medically important snakes of South Eastern Asia

Category 1:

- Agkistrodon Rhodostoma
- Echis Carinatus (Saw scaled viper)
- Enhydrina Schistosa
- Naja naja (Common cobra)
- Vipera Russell' s (Russell' s Viper)

Category 2:

- Bungarus Cerulurs (Common Krait)
- Hydrophis Cyanocinctus
- Hapemic Hardwicki
- Ophisthophagus Hannah (King Cobra)
- Trimeresurus Purpureomaculatus

Category 3:

- Trimeresurus Albolabris
- T. Wagleri

Venomous snakes of India:

- BIG FOUR - Saw Scaled Viper, Russell' s Viper, Common Cobra and Common Krait.

Saw Scaled Viper

- Zoological name: Echis Carinatus
- Tamil name: Surattai Pambu, Viriyan Pambu
- Smallest member of big four snakes

- 90cm in length. Life span - 2 to 5 years
- Head - short, wide, pear shaped. Snout - short and rounded
- Eyes - Relatively large and set well forward
- Body - moderately slender and cylindrical
- Dorsal scales - mostly ridged / keeled
- Has broad belly scales with brown or dark spots. World' s most dangerous snake because of highly toxic venom, abundance near cultivated areas, aggressive, easily excitable environment, most Echis species are oviparous.

Russell' s Viper:

- Zoological name: Vipera Russell' s or Daboia russelli
- Tamil name: Kannadi Virian
- Flat, heavy triangular head with a white v-shaped march, angle of V pointing forwards
- Has 3 rows of diamond shaped black or brown spots along the back waving white ringed edges

- Breeding - ovoviviparous.

Common Cobra:

- Tamil name: Nalla Pambu
- Hood is the most distinctive and impressive characteristic of Indian Cobra. There is a white band in the region where the hood touches the body. Spectacle masks on the rear side (monocellate / bicellate)
- Portion of neck surrounding the spectacle mark is darker than the rest of the back. Length : 6 to 8 feet ; Habitat : Found in plains, jungles, open fields ; Breeding - Oviparous.

King Cobra: (Ophiphagus)

- Large and powerful - Male snakes larger and thicker than female
- Length - 12 feet upto 20 feet
- Life span - 25 years
- Color - It has faint, pale yellow cross bands on the length of body
- Size larger than common Cobra
- No spectacle mark on hood
- Plain stripes on the neck may or may not be present
- Habitat: In forested areas
- Delivers a large quantity of highly potent venom in a single bite

Common Krait:

- Tamil name: Kattu viriyan
- Male lager than female and has a longer tail
- Creamy white belly. Average length - 1m
- Subcardial scales after the anal plate - not divided
- Large hexagonal scales - running down its spines
- Stripes are very narrow, single or double white lines
- Over the back, more prominent near the tail. Nocturnal in habit.

Banded Krait:

- Zoological name: Bungarus Fasciatus
- Tamil name: Kattu viriyan
- Alternate jet black and yellow cross bands each of equal size measuring about 5cm. Body cross section - Triangular. Marked vertebral ridge consisting of enlarged hexagonal vertebral scales. Head - Broad and depressed. Black mark on the neck, which is spread upto the eyes. Length - 1 to 2 m. Small tail.

Sea snakes:

- Found in warm coastal water from Indian ocean to the Pacific. Color: Black, greenish back or bluish black with or without bands.

Small eyes, prominent nostrils on the top of head, small tuberculated dorsal scales. Have paddle like tails and many have laterally compressed bodies that give them cell like appearance.

Difference between Venomous and non venomous snakes:

S. NO	TRAIT	VENOMOUS SNAKES	NON - VENOMOUS SNAKES
1.	General appearances	Stout, dull colored	Slender, brightly colored
2.	Head	Triangular	Rounded or oval
3.	Head scales	(1) Small (in vipers). large in others (2) large (a) pit between eye and nostril - pit viper (b) third supralabial touches the eye and nasal shields [cobra or coral snake](c)(i) No pit (ii) third supralabial does not touch the eye and nasal shields (iii) central row of scales on back enlarged and hexagonal in shape (iv) 4^{th} infralabial is the largest [Kraits]	Large with exceptions are mentioned in adjacent column

4.	Belly scaled	Large and cover entire breadth	Small, like those on the back. May be larger, but do not cover the entire breadth of belly double rows
5.	Anal plate and subcaudal scales	Single row	Double row
6.	Teeth	Two long fangs and a row of smaller teeth	Several small teeth arranged in rows, but no fangs
7.	Fangs	Canalized, like hypodermic needles [vipers]or have grooves [cobra]	Short and solid. Contain no canal or groove
8.	Poison glands	Present	Absent
9.	Saliva	Contains toxic polypeptides and enzymes	No
10.	Tall	(1) May be rounded or flattened (2) tapers abruptly	(1) Always rounded (2) tapers gradually
11.	Habits	Mainly nocturnal	Diurnal

Difference between Cobra and Viper:

S. NO	FEATURE	COBRA	VIPER
1.	Body and neck	Long and cylindrical	Short body; narrow neck
2.	Head	(1) Small (2) seldom broader than body (3) covered by large scales	(1) Large and triangular (due to the venom glands located in both temporal regions) (2) broader than body (3) covered by numerous small scales
3.	Pupil	Round	Vertical
4.	Upper jaw	Carries poison fangs and other teeth	Carries only poison fangs
5.	Fangs	Short, grooved, fixed	Long, canalized, mobile. Retract posteriorly when the mouthis closed

6.	Venom	Neurotoxic	Hemotoxic
7.	Tail	Round	Tapering
8.	Eggs	Lays eggs (oviparous)	Gives birth to young ones (viviparous)

Snake bite poisoning deaths are considered as unnatural deaths in India and hence post-mortem examination of deceased persons is mandatory according to law. As there is monetary assistance provided by Government of India to the deceased persons family in case of snake bite, there is high probability of maligning death due to other causes as Snake bite deaths, hence post-mortem examination in such cases may lead to proper deliver of justice. Ophitoxemia (Greek -Ophis - Serpent, Toxon -Poisoning) is poisoning by snake venom. In more than 50% of cases, inadequate venom is injected, producing mild symptoms. Signs and symptoms depends upon

1. Snake dependent factor

- Species and size of snake

- Condition of its fang and venom glands
- Pathogens present in snake venom
- Nature of bite - location, number and depth of bite
- Amount of venom injection

2. Victim dependent factors

- Age and built of victim
- Victim' s sensitivity to venom

3. Community dependent factors

- First aid and medical care given

Fatal dose and total yield of venom in various snakes

Snake	Fatal dose in terms of dried venom	Total yield in one bite in terms of dried venom
KRAIT	6 mg	20 mg
SAW SEALED VIPER	8 mg	25 mg
RUSSELL' S VIPER	20 mg	150-200 mg
COBRA	15 mg	200-350 mg

Fatal period

Cobra - 8 hrs

Krait - 18 hrs

Russell' s viper - 3 days

Saw scaled viper - 5 days

Viperine venom is mainly hemolytic and cause intravascular hemolysis. Though elapid bite is considered neurotoxic, all snake venom are mixed toxins and the type of toxins differ among the species.

Renal involvement in elapid bite may be due to the following reasons:

1. Intrinsic activity (direct cytotoxic effect) on kidneys.

2. Immunologically mediated nephropathy

3. Ischaemia induced nephropathy

Most common cause of death encountered in cobra bites is respiratory paralysis and in viperine bites is hemolysis and haemorrhage.

Snake venom and their relevant components

Snake venom are rich bio - resource of biologically active components, but only one percent of these molecules have been

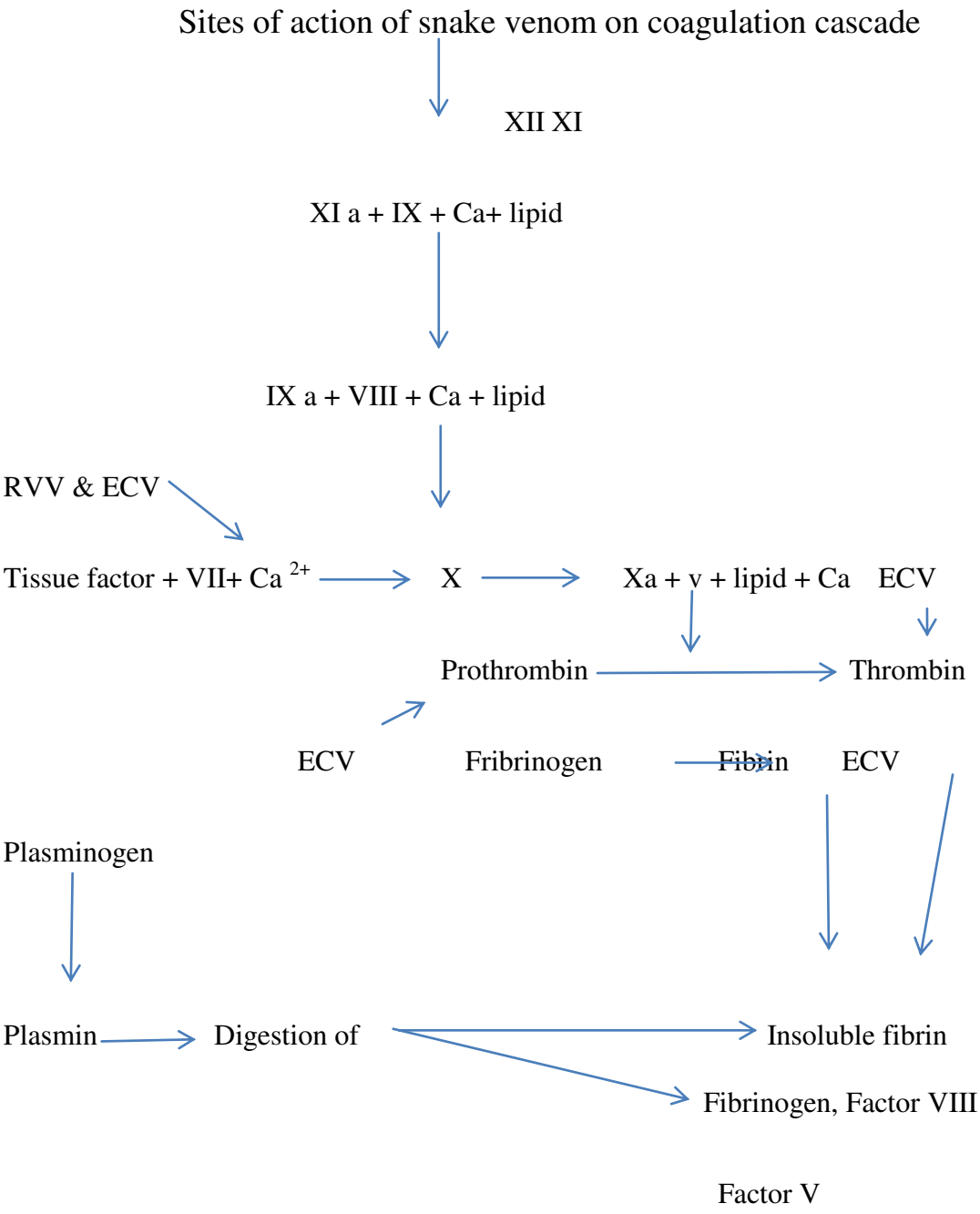
characterised. Identification and characterisation of toxic components present in snake venom are the main step not only to understand the pathophysiological changes observed after bites, but also to improve the treatment after snake bites. Snake venom is composed of 90% of proteins, polypeptides, many other organic and inorganic substances. Most snakes have 6-12% of enzymes in their venom. No venomous snake has all components.

Components of snake venom are:

- Adenosine tri-phosphate (ATPase) - Cause shock and immobilization of smaller prey.
- Agglutinins- RBC agglutination.
- Amino acid oxidase - Digestion and triggering of enzymes.
- Biological amines eg. Histamine, Serotonin - Pain and increased permeability.
- Cardiotoxin - Toxic to heart, affects skeletal and smooth muscles, found mainly in Cobra.
- Cholinesterase - Muscle paralysant, found mainly in elapidae.
- Cytolysin - Lysis of all cell walls found mainly in Viper.
- Fibrin ferments - Increase coagulation process
- Haemorrhage - Zinc containing metalloproteases.
- Hyaluronidase

- Hydrolases
- Neurotoxins
- Nucleases
- Lecithinases
- Ophanin - Belongs to Cysteine rich secretory protein family. Inhibits voltage dependent ca^{2+} channels
- Ophioxidase,
- Peptides and polypeptides
- Phosphodiesterase
- Phospholipase A,B,C,D
- Phosphotidases
- Proteases
- Proteinase
- Proteolytic enzymes

Snake venom travel through lymphatics and superficial veins and are poisonous only when injected and harmless per oral. Excretion is through kidneys, milk, salivary glands and mucus membranes.



Pathogenesis of Renal lesions

Acute Kidney Injury (AKI) is caused mainly from venom of Viperid bites, also seen in elapid bites also. Independent risk factors predicting AKI are cellulitis, Regional lymphadenopathy, Gangrene at the bite area, marked abdominal pain, myalgias, hypotension,

consumption coagulopathy, increased bite to needle time (i.e) time taken to administer antisnake venom after snake bite, intravascular hemolysis.

Hypotension and circulatory collapse set in motion a chain of hemodynamic disturbances, which are known to culminate in ischaemic acute renal failure. Another factor of pathogenetic significance in snake bite induced acute renal failure is intravascular hemolysis. Hemolysis results from action of phospholipase A_2 and a basic protein called " direct lytic factor" . phospholipase A_2 causes hemolysis by direct hydrolysis of red cell membrance phospholipids or indirectly via production of strongly hemolytic lysolecithin. Microangiopathic hemolytic anemia has been demonstrated.

Snake venom induced consumption coagulopathy also plays a major pathogenetic role in the renal lesions of snake bite induced cortical necrosis. Presence of fibrin thrombi in renal microvasculature and in glomerular capillaries, findings of microangiopathic hemolytic anemia, thrombocytopenia in patients with snake bite induced cortical necrosis strongly support the causogenic effect of AKI by coagulopathy. Mild to moderate transitory proteinuria in patients of snake bite indicates that hypersensitivity to venoms play a role in renal failure.

Role of complement in the causation of renal damage is far from clear. Serum complement C_3 is severely depressed by administration of

Viper venom in experimental animals. Evidence of complement activation chiefly via alternate pathway, has been obtained by Russell' s Viper, Saw scaled viper and Bloomslang.

Cross examination of kidneys in snake bite cases shows a slightly edematous pale appearance with cortices pale and medulla congested with a sharp demarcation at the corticomedullary junction. Acute tubular necrosis is the most significant histopathological change noted, other lesions observed are cortical necrosis, glomerular lesions and interstitial lesions. Findings noted in acute tubular necrosis were

1. Tubular dilated and lined by flattened epithelium.

2. Cloudy degeneration of tubular epithelium along with frank necrosis and desquamation of necrotic cells from basement membrane into lumen.

3. Interstitial edema, haemorrhage and inflammatory cell infiltration.

Findings present in case of acute cortical necrosis

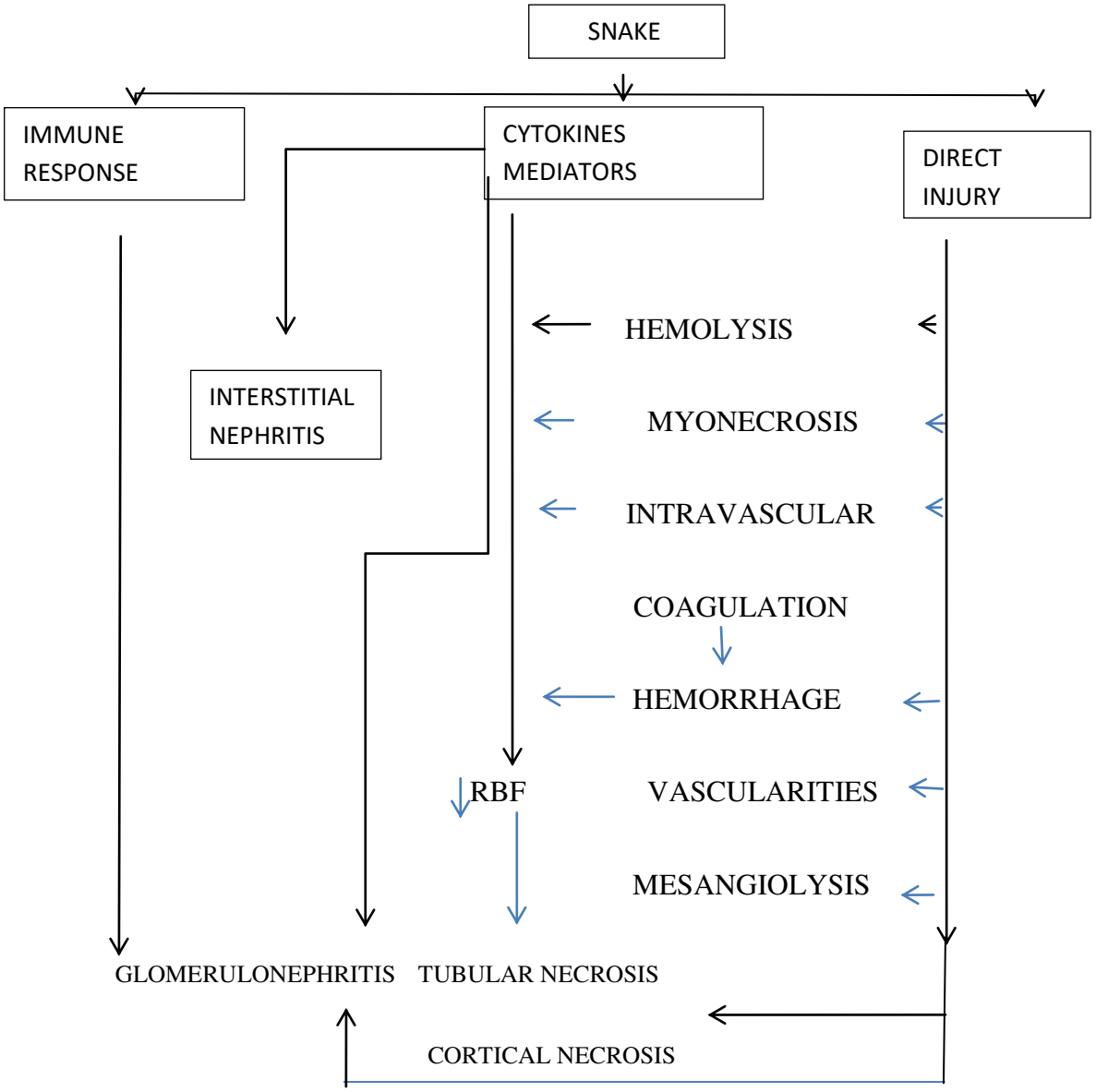
1. Fibrin thrombi in arterioles

2. Patchy necrosis of glomerular and tubular elements.

Other most significant histopathological changes noted are proliferative glomerulonephritis, mild mesangial proliferation, diffuse glomerular

hypercellularity, ballooning dilatation of capillary loops in glomerulus, endothelial cell swelling, and glomerular hypercellularity. Cytotoxin effect of venom on the kidney is considered to play a major role in pathogenesis of ARF.

Pathogenesis of nephropathy in snake bite:



Clinicopathological correlation:

PATHOLOGY	CLINICAL RENAL MANIFESTATION
Mesangiolysis	Normal renal function, normal urine finding, or haematuria
Mesangial proliferative glomerulonephritis	Normal renal function, normal urine finding or haematuria, or mild proteinuria. Occasional heavy proteinuria with complete resolution
Diffuse proliferative glomerulonephritis	Mild renal failure, haematuria, proteinuria
Extracapillary proliferative glomerulonephritis(usually associated with tubular necrosis)	Severe renal failure and haematuria with prolonged clinical course
Vasculitis (usually associated with tubular necrosis)	Severe renal failure with prolonged clinical course
Tubular necrosis	Acute renal failure
Acute diffuse interstitial nephritis (usually associated with tubular necrosis)	Severe renal failure with prolonged clinical course
Cortical necrosis	Severe acute renal failure with residual damage or without recovery

2. AIM AND OBJECTIVES

1. To know the epidemiological profile of snake bite cases.
2. To know the spectrum of histopathological changes of kidney in snake bite cases autopsied at Madurai Medical College & Hospital.
3. To analyse and compare the renal lesions among different snake bites post mortem and its association with mortality.
4. To find out other major causes of death in snake bite.

3. REVIEW OF LITERATURE

Sitpriya and Boonpucknavig demonstrated the renal lesion in snake bite cases after studying the renal biopsy of 31 patients bitten by tropical poisonous snakes. Described that extra capillary glomerulo nephritis associated with acute tubular necrosis were demonstrated in patients after they are envenomated by Russell' s viper and Greenpit viper. There are some of unique features that induces ARF are severe tubular and vascular lesions, increased apoptosis in distal tubules and the presence of eosinophils, mast cells and hyperblastic fibroblast in the intestitium.

KS Chug in his study on snake bite induced renal failure observed that a combination of mechanisms including bleeding, hypotension, circulatory collapse, intravascular hemolysis, and disseminated intravascular coagulation with or without microangiopathy was responsible for the development of acute renal failure following snake bite.

Proliferative glomerulonephritis due to toxic action of the venom was reported by Seedat.et.al.

Sant & Perandare and Tembe and Sant described toxic proliferative glomerulites with proliferation and swelling of endothelial cells. In their experimental studies (rabbits and Bonnet monkeys) injection of Echis carinatus venom produce endothelial swelling and proliferation.

Steinbeck described nephrotic syndrome as the cause of renal failure in snake bite cases. Ischemia induced nephropathy has also been reported from neurotoxic snake bites. The most common finding in renal his to pathology was acute tubular necrosis followed by cortical necrosis.

Cattell and Bradfield noted that the earliest lesion is the appearance of loose platelet aggregates in glomerular cappilaries. Features unique to snake bite induced ARF that differentiated it from other causes of ARF are:

Severe tubular and vascular lesions, increased apoptosis in distal tubules, and the presence of esinophils, mast cell and hyperplastic fibroblast in the interstitium.

Bilateral cortical necrosis of the kidneys was first described by Juhel-Renoy (1886). The lesion is characterised by an acute necrosis of large portions of renal cortices. The necrosis is ischaemic in origin.

Reid and Jenkins(1984) showed that elapid venom (cobra) was capable of releasing renin by acting directly on the kidneys of adrenalectomised rats.

Amorim & Mello (1954) demonstrated distal tubule necrosis in rattle snake envenomation.

Oram.et.al (1963) demonstrated calcitic material, shrunken glomeruli with thick capsules, dense infiltrate of mononuclear cells in Echis carinarus envenomation.

Azevedo & Teixeira suggested the presence of glomerulo nephritis in cobra bite.

Neelakanthi Ratnatunga et al demonstrated the occurrence of chronic interstitial inflammation after AKI in snake bite cases, thus suggesting the possibility of continuing renal damage by the process of interstitial inflammation and morosis mediated by activated CD_3 lymphocyte.

Nanayakkara et al indicated inflammatory infiltration, glomerular congestion, hemorrhages and focal necrosis in krait envenomation.

Mittal.B.V (J. Post grad Med 1994) depicted tubular necrosis (53.6%), cortical necrosis (24.3%) as the main causes of ARF. Tubular degeneration in the form of cloudy or vascular change in the absence of

frank tubular necrosis was demonstrated in fewer cases. Glomerular changes noted in the study are ballooning, dilated glomerular capillary loops, mesangiolysis, endothelial cell swelling and splitting of glomerular basement membrane.

Striker et al scored the glomerular and tubular changes according to the severity present.

Galay et al reported tubular injury in 4/5 patients in addition to diffuse interstitial infiltrates. This study also demonstrated the chronic kidney disease/end stage renal disease that follows AKI.

Mukhopadhyay et al demonstrated the incidence, pattern and spectrum of renal lesion in snake bite by Indian cobra. The major renal changes found during histology were tubular necrosis, cortical necrosis, interstitial nephritis. Glomerular changes (glomerular collapse and fibrin deposits) were seen in 2 cases of fatal cobra bite.

Toru Hifumi et al snake bite are the most life threatening injuries which requires intensive care. In this study, bites involved mamushi (Japanese pit viper), habu (Japanese pit viper), and yamakagashi snakes (colubrid snakes) which are the Japan and other Asian countries inhabitant snakes were studied. Mamushi causes swelling and the pain starts to spread slowly from the site of bite. Which results in the

decrease platelet counts because of the platelet aggregation. Yamakagashi bites causes hemorrhagic symptoms and severe intravascular coagulation with fibrillation and turns to be the life threatening bite.

Kirpal S et al states that the ARF following the snake bite and also the effects of viperide venoms on the structure of renal and its function are studied in 157 cases where they found that 47 cases developed the history of ARF, 35 cases are studied histopathologically, 45 cases are given antibiotics and 8 received anti-snake venom. This study even stated that 10 cases had bilateral renal cortical necrosis, 8 had died and 23 patients had severe acute tubular lesions in which 4 had died.

Varagunam T and Panabokke RG found that bilateral necrosis of the kidney is the most rare lesion of pathology. It follows some pregnancy complications like eclampsia. One such case has reported in this study. A 30 year old male has reported for not passing the urine for 16 days, later he died. The autopsy of the patient revealed that lesion was seen in the patient who died of uraemia after snake bite. Calcification of both renal cortices in patient has recovered from anuria after the snake bite. This occured due to the bilateral cortical necrosis of the kidneys after the bite.

Rajesh Waikhom et al in his study noted that snake bite can cause acute kidney injury through number of mechanisms. Many undergo renal replacement therapy. 60 patients developed dialysis - requiring severe AKI after snake bite and survived. Totally 25 patients (41%) shows renal involvement persistently as renal dysfunction, proteinuria, or hypertension for a period of 45 months. 5% progressed to end stage renal disease and the remaining 20% had glomerular filtration rate < 45 mL/min.

The clinical and histopathological characteristics of patients were identified in the patients who developed acute interstitial nephritis following the snake envenomation. As the retrospective analysis of 88 patients who are diagnosed with snake envenomation - induced by acute interstitial nephritis are studied to know the further perceptions. For 7 patients Biopsies were performed because of the non-recovery of the kidney function, 5 patients are with acute interstitial nephritis (5.7%) . Priyamvada PS et al concluded that all the patients admitted had severe envenomation and prolonged renal failure. So, the biopsy showed to have mixed infiltrates like lymphocytes, with variable proportions of eosinophils, neutrophils and plasma cells. The corticosteroid response was only 80%.

Mrudul V Dharod et al evaluated the clinical profile of snake bite patients and determined the predictors who are developing AKI following the snake bite. In this study 281 patients with snake envenomation were considered. 87 developed AKI and 194 did not develop. Among 87 patients 61 (70.11%) were male and in 194 patients 117 (60.30%) were male. Out of 281 patients, 232 had cellulitis, 113 with bleeding tendencies, 87 with oliguria, 76 with neuroparalysis and 23 with hypotension.

Kohli HS and Sakhuja V in their article snake bites and acute renal failure noted acute tubular necrosis as the predominant renal lesion in patients of snake bite induced renal failure other lesions include diffuse or patchy cortical necrosis.

Acute renal failure is the important cause of morbidity and mortality in snake bite patients, especially for tropical countries. Tushar B Patil et al studied 57 patients of snake bite induced ARF, and the patients who required dialysis were also included. All the patients were treated with anti snake venom. Prevalence of ARF in snake bite was 20.48% and the common clinical manifestations were cellulitis, oliguria, edema, hematuria, altered sensorium and bleeding.

Fabia MO et al demonstarted that one hundred cases of Crotalusdurissus bite were studied in which 29 patients developed ARF.

Of those, 24% required dialysis and 10% died. C.durissus venom-induced ARF had high prevalence (29%) and it is related to significant mortality in young and healthy patients. A delay in the administration of specific antivenom, presence of CK>2000 U/L, and age <12 years were independent risk factors for ARF. Diuresis >90 mL/hr at admission was a protective factor.

According to Adrija Hajira et al Viper venoms acts as hemotoxic. Snake bites depends on certain toxins that constitutes venom. To reduce the morbidity and mortality it is important to notify that antiserum is administered at appropriate dose and given to the patient as early as possible after the snake bite. A 30 year old male farmer was a victim in this case who got viper bite while working in his field. Due to some inevitable situation the ASV was given 5 hours delayed. Due to this the patient developed severe epigastric pain and vomiting. Ultrasound of the abdomen showed the pancreas is bulky and free fluid is seen in intraperitoneal space. These cases are unique only because of their extensive tissue necrosis.

Medically snakes have fangs at the front of their mouths which is used to inject the venom. According to H. Alistair Reid, the poisonous snakes like elapid have short fangs, vipers have long erectile fangs. The distinction of poisonous from non-poisonous snakes is often difficult,

but it is not important for the clinicians. Epidemiological studies have confirmed that snake bites in the tropic and in the rural areas is a hazard. Mostly bites occur in day light and on the foot of the victim and the severity of the poisoning is not related to the time of the bite, breeding habits of the snake or the age of the victims.

A study conducted by Ali G and colleagues recorded the occurrence of acute renal failure in 62(17%) patients amongst 360 patients with saw scaled viper envenomation.

A study by Athappan G et al on 1548 patients of snake bite showed that 159(13.5%) patients developed acute renal failure.

Suchithra N, Pappachan JM conducted a study in kerala in May 2005-Dec 2006. Total of 586 cases were studied the results showed 34% (200 cases) were envenoming. Regional lymphadenitis occured in 61% mortality rate was 3%, 39.5% had complication with acute renal failure. Higher complication rates were seen in those with coagulopathy, leucocytosis and those with the patients, who received late anti snake venom. The study proved early administration of ASV reduces risk of complications. The presence of leucocytosis and severe coagulopathy can predict adverse outcomes.

Atif Sitwat Hayat et al, conducted a study in Hyderabad where 100 cases were studied and concluded that 57 viper bites which presented with haemostatic abnormalities and 35 were elapid bites which presented with neuroparalytic symptoms, fang marks noted in 90% cases. Among these 100 cases one patient had ARF and another patient had DIC. 3% cases of elapid bites needed assisted ventilation. 4 patients had adverse affects after Antivenom. Overall mortality was 4%.

Bawaskar et al at a study done in Maharashtra, India 182 cases of snake bite were studied, out of these 55 (30.2%) were echiscarinatus, 38 (20.8%) were Russell' s viper, 48 (26.3%) were Krait and 41 (22.5%) were cobra.

Emam SJ, Nikzamir in a study conducted in 103 patients, concluded that 72 of them were males 27 were females. 50.5% were found to have hemoglubinuria, 40.9% had proteinuria, 29.1% had bactiuria, 33% had anemia, 74.8% had rhabdomyolysis, 45.6% had myoglobinuria, 12.7% had leucocytosis, 1.9% had thrombocytopenia, 65% had coagulopathy.

Mohammed Al-Homrany in a study conducted in an adult patient stated with a history of snake bite who was administered with antivenom and adequate hydration had developed acute renal failure secondary to

dissemination of intravascular coagulation and rhabdomyolysis. In this case the clinical course is highly subjected to acute tubular necrosis.

Acute renal failure is an important cause of morbidity and mortality in snake bite patients, especially in tropical countries. This study was conducted by TB Patil, Yv Bansod with an aim to describe the clinical profile and to identify predictors of poor outcome in snake bite induced acute renal failure. They have even included 57 patients who require renal dialysis were also included. All patients received standard treatment including anti-snake venom. Some patients even received peritoneal or hemodialysis which depends on the availability of resources. Prevalence of ARF in snake bite was 20.48%. Common clinical manifestations were local cellulitis (100%), oliguria (84.2%), edema (33.3%), hematuria (29.8%), altered sensorium (26.3%) and bleeding manifestations (22.8%). Common laboratory findings were albuminuria (100%), anemia (54.3%), leucocytosis (61.4%), thrombocytopenia (42.1%), coagulopathy (36.8%), metabolic acidosis (31.5%).

Rubina Naqvi described the clinical spectrum and outcome of patients presenting to a tertiary care kidney center, developing acute kidney injury after snake bite. As it is an observational study, patients with snake bite are included. AKI is defined as the criteria with sudden

rise in creatinine or decline in urine output or both. During the study period 115 cases of AKI were registered. Medium range of age of the patients are between 6-70 years. Snake bite leading to multiple complications including renal failure and death, is a major health issue in tropical countries.

Anjana Silva, Rivikelum Samarasinghe et al stated that Sri Lanka has high snake bite related morbidity with 37000 hospital admissions annually. The highest snake bite incidence, mortality and case fatality rates are recorded in the dry zone, particularly in the North Central Province where farms, paddy fields and jungles are in close proximity. Russell' s viper will cause 50 % of all snake bites in the dry zone, most of them result in life threatening systemic envenomation characterized by coagulopathy, acute renal failure and neurological involvement. Pathological findings included progressive tubulointerstitial nephropathy, which indicates environmental or occupational agents. Due to the persistent inability to find a cause for it, this disease was called CKD of unknown etiology.

Sant SM, Purandare NM stated that an average 150 patients with snake bite were studied, among these 48 necropsies was identified as these cases forms the basis. In remaining patients lesions like proliferative glomerulitis, cortical necrosis, interstitial haemorrhagic

nephritis, focal necrosis of liver, massive haemorrhage in the brain, toxic myocarditis, lung oedema, lung haemorrhage are detected.

Muhamed qamar ali stated that snake bite is predominantly an occupational hazard in the tropical countries. Agriculture remains the principal means of livelihood for 58.4 % of India' s population and for a considerable proportion of the population risk of snake bite. Acute renal failure can occur with the bite of any venomous snake and it is more common with snakes of the viperine species. The incidence of the acute renal failure in India is 13-32 % following the viper bite.

Naren Sarkar, Souvik Basu, Preeti Chandra, Soumeek Chowdhuri and Partha Pratim Mukopadhyay suggested that death due to poisonous snakebite is a formidable health hazard. It is especially in agrarian countries. In this study three cases of deaths in consecutive bites by a single banded Krait was considered. Significant renal involvement was also found during autopsy. The kidneys showed interstitial haemorrhage and inflammatory cell infiltration. The renal changes were similar in all the three cases bitten by the same snake.

Christopher Kruk et al stated that the brown snake bite envenomation resulted in the caogulopathy, thrombocytopenia, anemia and acute renal failure. Coagulopathy and thrombocytopenia are well recognised complications of brown snake bite. However the acute renal

failure from brown snake bite has been described on only six occasions in the Australian literature. The renal failure was self - limiting in both cases though one patient required dialysis for 12 days. Neither patient developed rhabdomyolysis.

According to M Pal et al histopathological studies conducted showed some changes which revealed that acute tubular necrosis and cortical necrosis are the predominant findings along with glomerular changes in few cases. Similarly, other findings are thrombosis, diffuse, focal and patchy necrosis, balloning dialtions, fibrin thrombosis, etc.

Prasad et al found major variations in the phospholipases A2 in the venom samples of India, toxic PLA2 activity showed a dramatic decrease as one moves towards eastern regions of India.

I F Inamdar et al demonstrated that the cause of case - fatality rate is 14.8% (30/203), most common cause of mortality in our study was coagulopathy 42.2% (19/45) followed by ARF 28.4% (26/88), neuroparalysis 20.5% (8/39) and cellulitis 15.8% (29/184). Mortality rates in other studies varied from 3-11%. Apparently high mortality rate in our study may be due to delay in arriving at the hospital after the snake bite or external factors which increase the chances of mortality are not receiving first aid, unavailability of anti-snake venom (ASV)

Butt KZ et al in his study notified that leukocytosis was most common followed by WBCT > 20 minute, raised SGPT and INR >1.5. Significantly higher mortality rate is observed in cases with WBCT.

Myo-Khin et al in his study noted haemostatic disturbances are known to be the pathological mechanism causing fulminant disease in hematoxic snake bite and bleeding tendency is well recognized as an indicator of greater risk of mortality. Most of vasculotoxic bites were associated with cellulitis (90.60%) and coagulopathy (19%).

Bhat RN et al and Saini et al in their study recorded haemorrhagic manifestations in 65% cases.

In the study conducted by Dhannya et al, the haemorrhagic manifestations was recorded in 76% cases and neuroparalysis was encountered in 19.20% cases. The common symptoms among these two manifestations were ptosis and blurring of vision in patients of which 7.4% cases developed respiratory failure. Acute renal failure was observed in 43.3% cases.

Incidence of acute renal failure in 14.4% cases was noted by Halesha BR et al. The dose of ASV was studied and found that dose of ASV was more in mortality group of patient as compare to survived group which was not statistically significant.

Meldrum et al stated that muscular weakness is a prominent feature in many of the poisonings by the bites of the Indian Cobra (Naja naja). Paralysis lead by asphyxia is the principal cause of death observed in experimental animals. Cobra venom produces a progressive diminution toxin which is responsible for the paralysis. It acts both at the neuromuscular junction and on the muscle cell membrane.

Nakai et al demonstrated that snakes belong to the family Elapidae possess potent neurotoxins in nature. The Indian cobra belongs to the same family of snakes but it differs from other neurotoxins in their amino acid composition and also in their pharmacological properties. Among various proteins " Toxin A" is isolated and purified by chromatography on sephadex G-75, G-50 and carboxymethylcellulose. Toxin A contains 8 cystine residues and it lack methionine like other snakes and scorpion. Analysis showed that N-terminal region of toxin A consists of isoleucine and C terminal amino acid is found to be alanine

Fatehyab Ali et al concluded that Russell' s viper venom cause serious local tissue and internal organ damage by means of lipid peroxidation which involves the direct reaction of oxygen and lipid to form free radicals. Another report states that increase in polyunsaturated fatty acids during envenomation may lead to increase in the rate of lipid

peroxidation resulting in tissue damage.Even a direct lipolytic action of the venom may be due to increasing cyclic AMP concentration. Free fatty acid liberation increased during envenomation will results in increase in the level of acetyl COA, leading to increase in the synthesis of cholesterol

Kasturiratne et al reported that snake envenoming is considered as major clinical issue in many tropical and subtropical countries. It was estimated 94,000 mortality every year in 1,841,000 envenoming cases globally. The highest cases where reported in south Asia, Southeast Asia and Sub Saharan Africa. The most complete data points, that envenomed bite constitute 18% and 30% of the total in India and Pakistan. Accuracy of estimating the total snakebite and mortality leads by envenoming become very difficult since majority of bite occur in rural areas of poor countries

4. MATERIALS AND METHODS

Data will be collected from the medico-legal autopsies of snake bite cases conducted at the Mortuary of Madurai Medical College & Hospital, Madurai from the period of April 2017 to March 2018.

Macroscopic and microscopic studies of kidneys to be done. Dissected kidneys to be sectioned, stained with hematoxylin and eosin stain and histological examination done under light microscope. Ideal section thickness for examination : 2 - 3 microns.

INCLUSION CRITERIA

Cases of autopsy done at Mortuary, Madurai Medical College & Hospital, Madurai with alleged history of snake bite during the period of April 2017 to March 2018.

EXCLUSION CRITERIA

1. Bite from other reptiles or animals.

2. Obscure histories are excluded.

3. Decomposed bodies.

4. Patients with pre-existing chronic renal illness

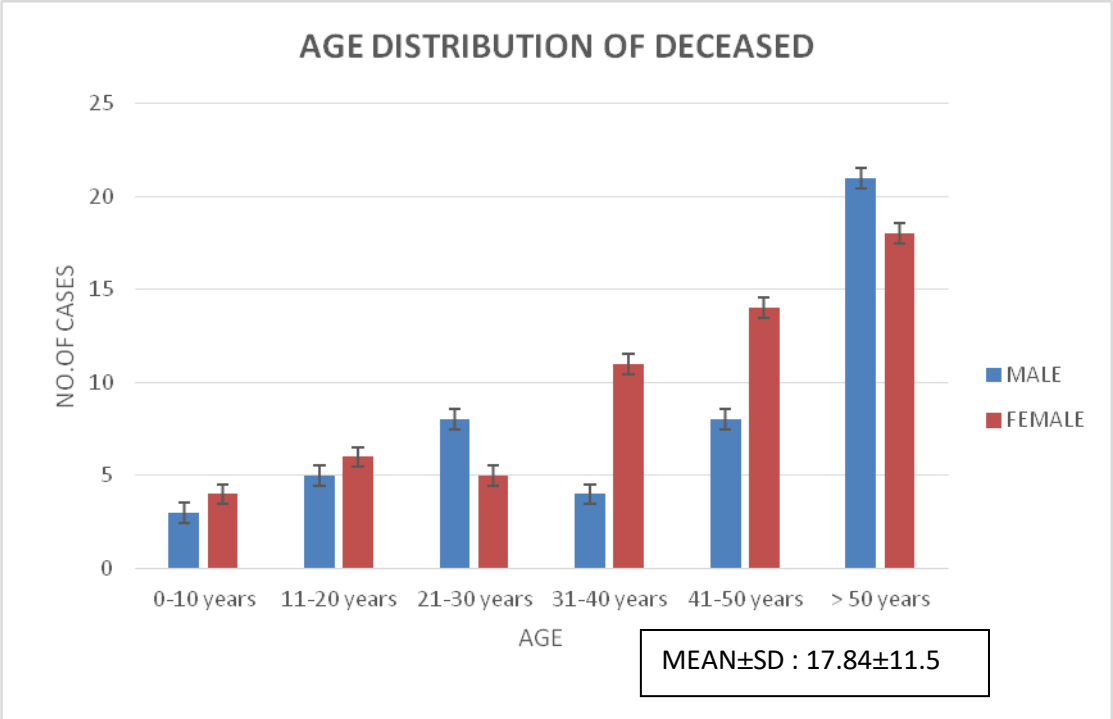
STATISTICAL ANALYSIS

The statistical software Graphpad Instat Version 3.06. The duplicates obtained were calculated and the Mean±SD were obtained for the values. The P value is found to be significant.

5. RESULTS

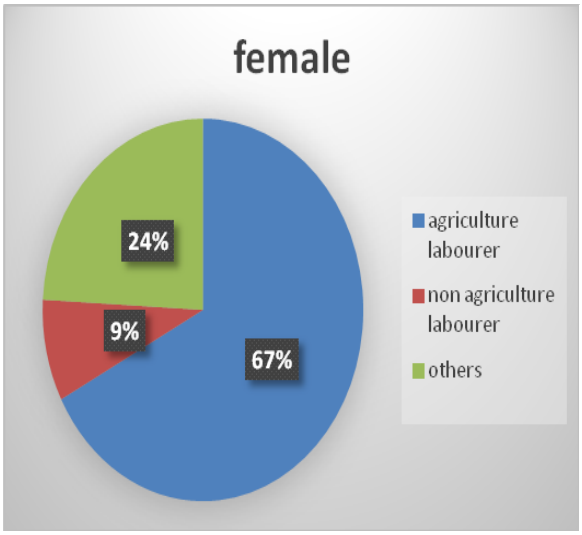
1. AGE DISTRIBUTION OF DECEASED

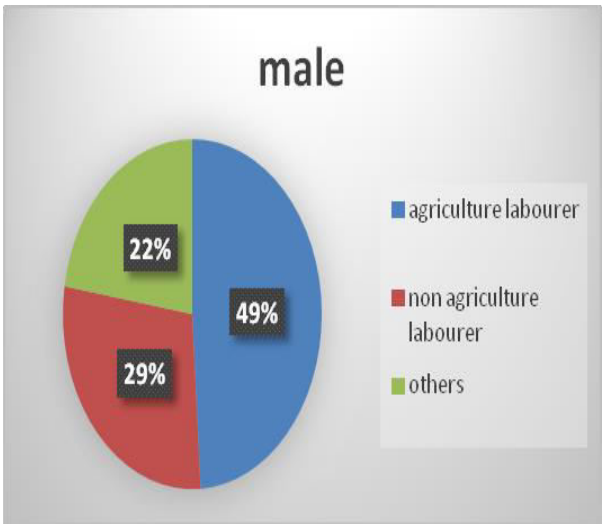
Sex	0-10 years	11-20 years	21-30 years	31-40 years	41-50 years	> 50 years
Male	3	5	8	4	8	21
Female	4	6	5	11	14	18
Total(n)	7	11	13	15	22	39
Percentage (%)	7	10	12	14	21	36



2. OCCUPATION PATTERN OF DECEASED

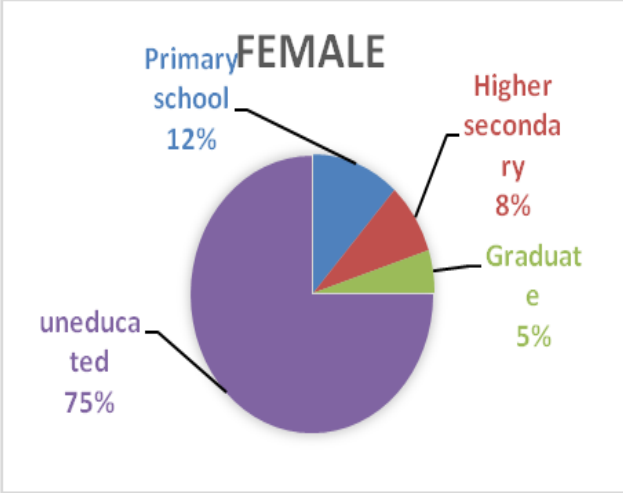
Occupation	Agriculture labourer	Non agriculture labourer	Others
Male	24	14	11
Female	39	5	14
Total (n)	63	19	25
Percentage (%)	59	18	23

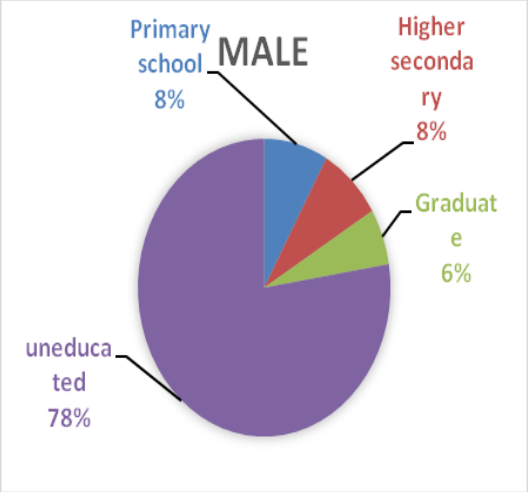




3. EDUCATION

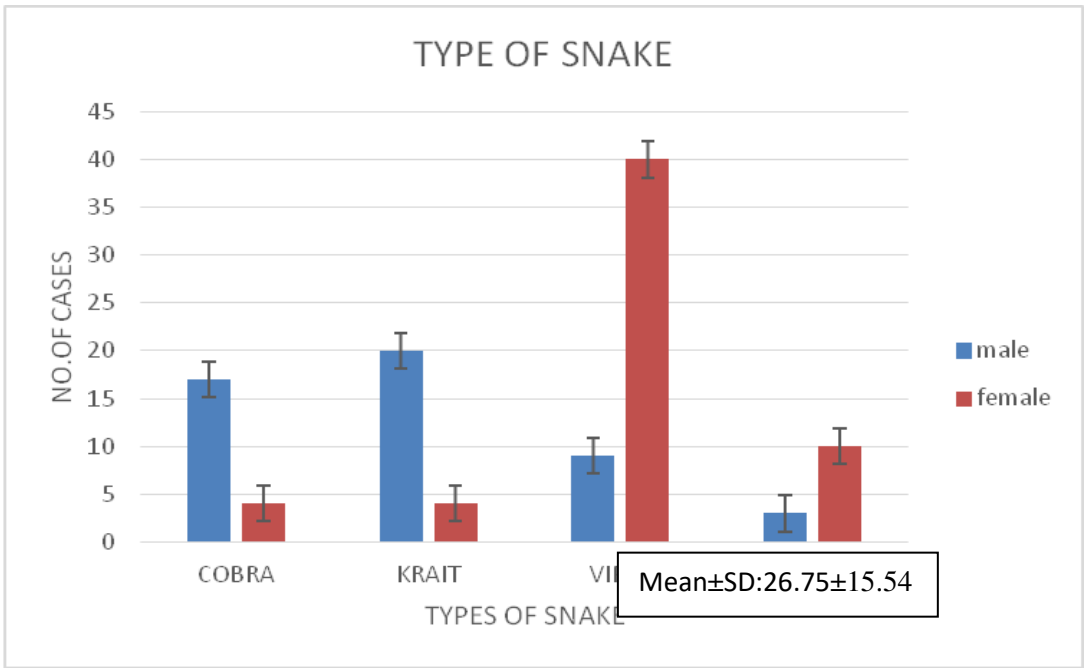
Education	Primary school	Higher secondary	Graduate	uneducated
Male	4	4	3	38
Female	7	5	3	45
Total (n)	11	9	6	83
Percentage (%)	10	8	6	76





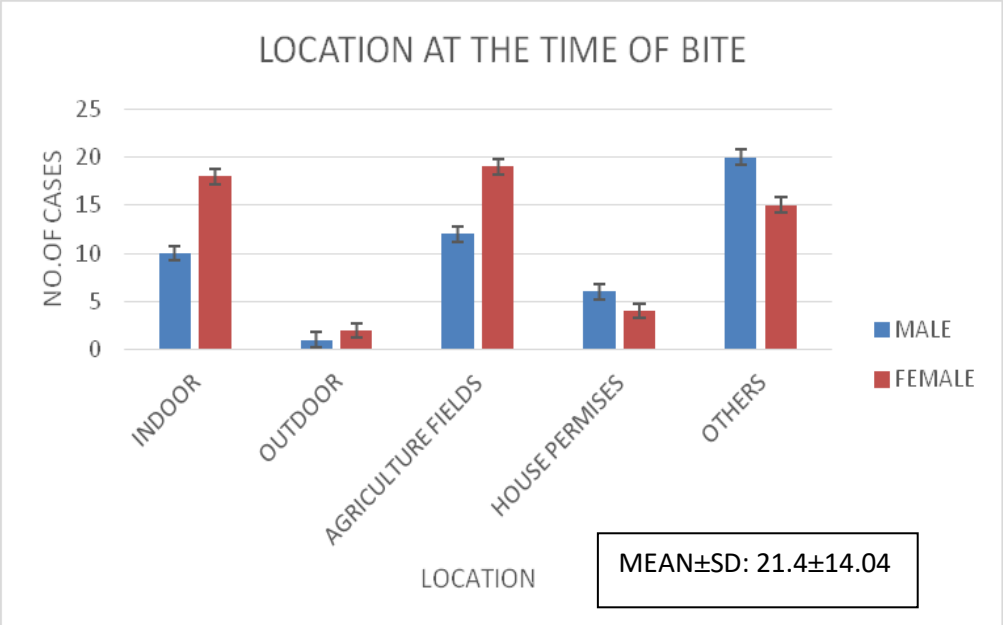
4. TYPE OF SNAKE

	No.of cases			
Type	Male	Female	Total (n)	Percentage (%)
Cobra	17	4	21	20
Krait	20	4	24	22
Viper	9	40	49	46
Undetermined	3	10	13	12



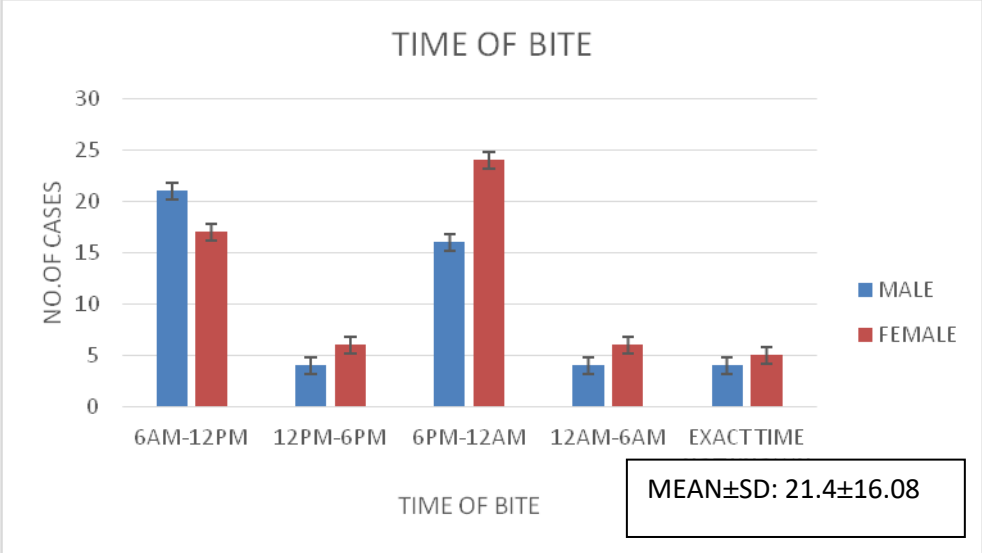
5. LOCATION AT THE TIME OF BITE

	No.of cases			
Location	Male	Female	Total (n)	Percentage (%)
Indoor	10	18	28	26
Outdoor	1	2	3	3
Agriculture field	12	19	31	29
House premises	6	4	10	9
Others	20	15	35	33



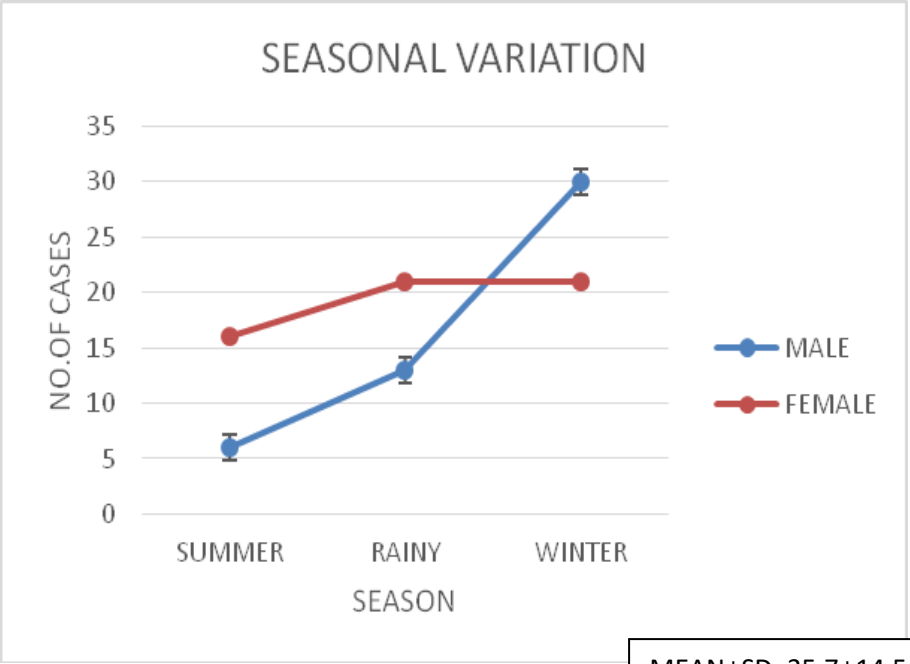
6. TIME OF BITE

	No.of cases			
Time of bite	Male	Female	Total (n)	Percentage (%)
6AM-12PM	21	17	38	36
12PM-6PM	4	6	10	9
6PM-12AM	16	24	40	37
12AM-6AM	4	6	10	9
Exact time not known	4	5	9	9



7. SEASONAL VARIATION

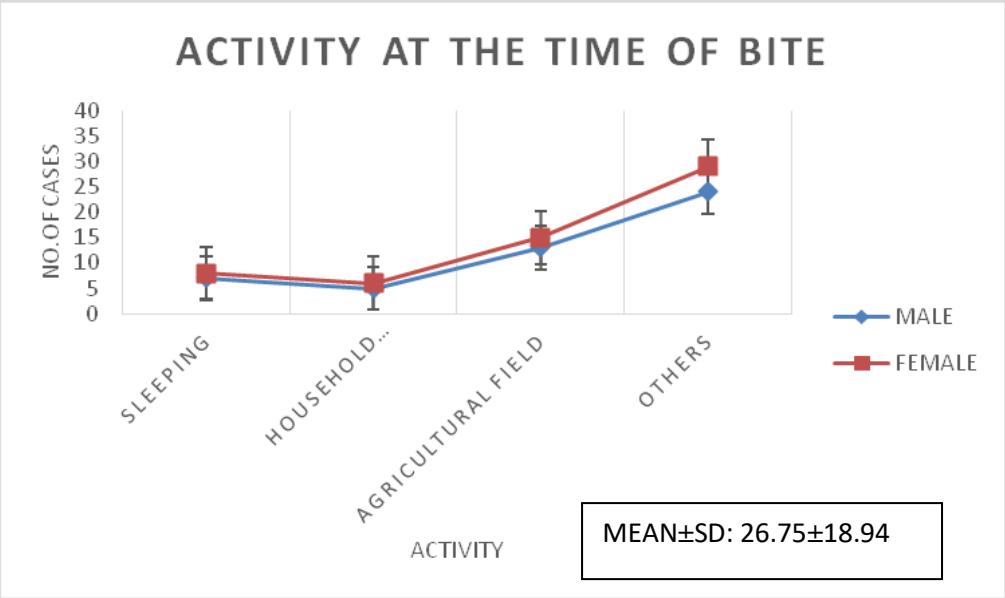
	No.of cases			
Season	Male	Female	Total (n)	Percentage (%)
Summer	6	16	22	20
Rainy	13	21	34	32
Winter	30	21	51	48



MEAN±SD: 35.7±14.5

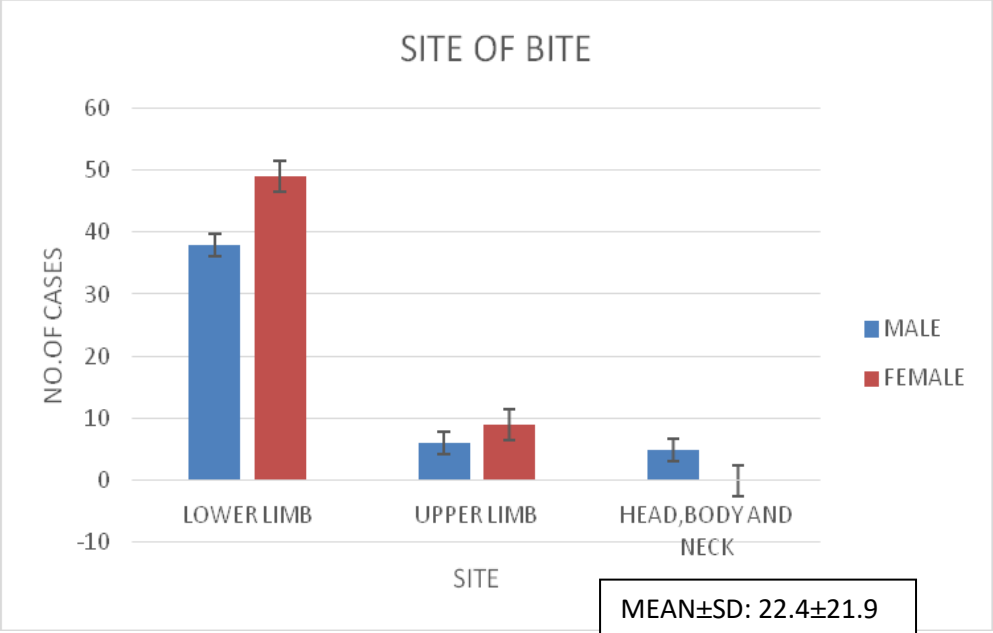
8. ACTIVITY AT THE TIME OF BITE

	No.of cases			
Activity	Male	Female	Total (n)	Percentage (%)
Sleeping	7	8	15	14
Household activities	5	6	11	10
Agricultural field	13	15	28	26
Others	24	29	53	50



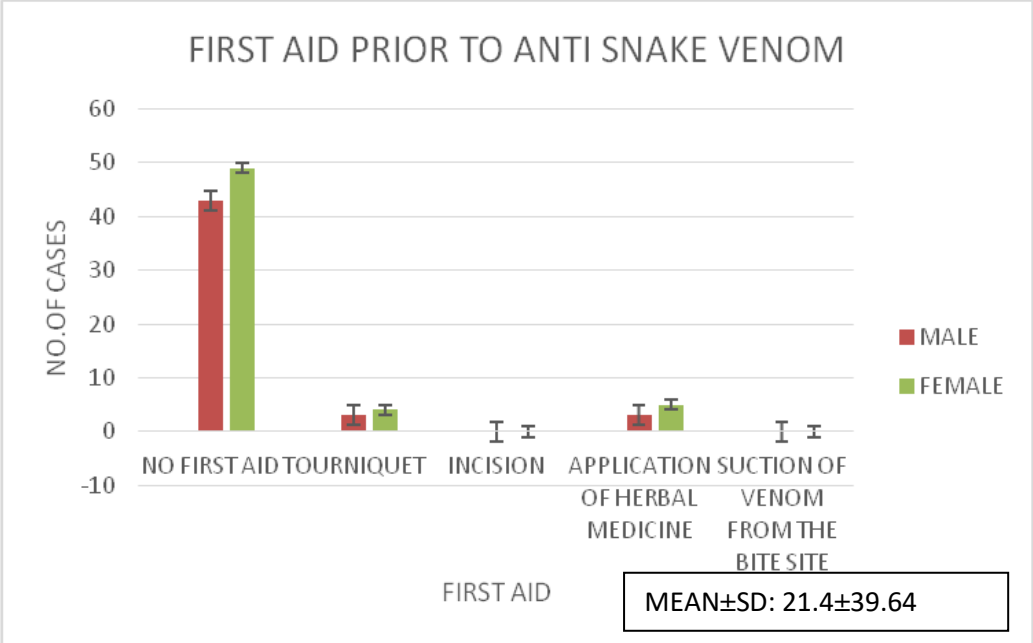
9. SITE OF BITE

	No.of cases			
Site	Male	Female	Total (n)	Percentage (%)
Lower limb	38	49	47	70
Upper limb	6	9	15	22
Head, body and neck	5	0	5	8



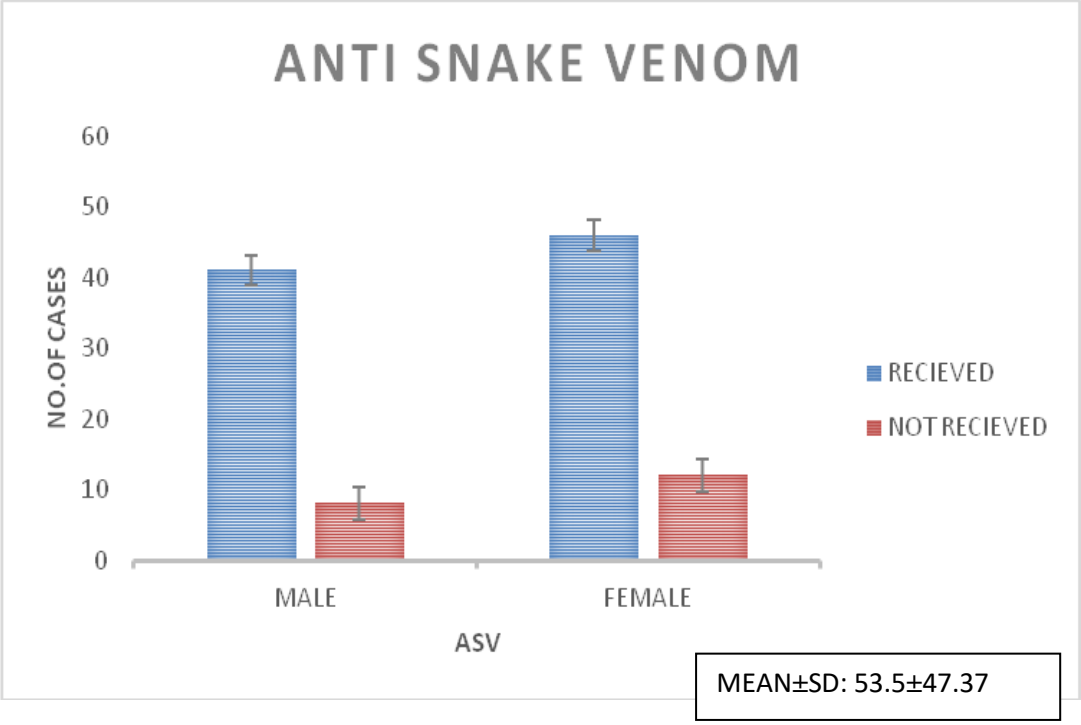
10. FIRST AID PRIOR TO ANTI SNAKE VENOM

	No.of cases			
First aid	Male	Female	Total (n)	Percentage (%)
No first aid	43	49	92	86
Tourniquet	3	4	7	7
Incision	0	0	0	0
Application of herbal medicine	3	5	8	7
Suction of venom form the bite site	0	0	0	0



11. ANTI SNAKE VENOM

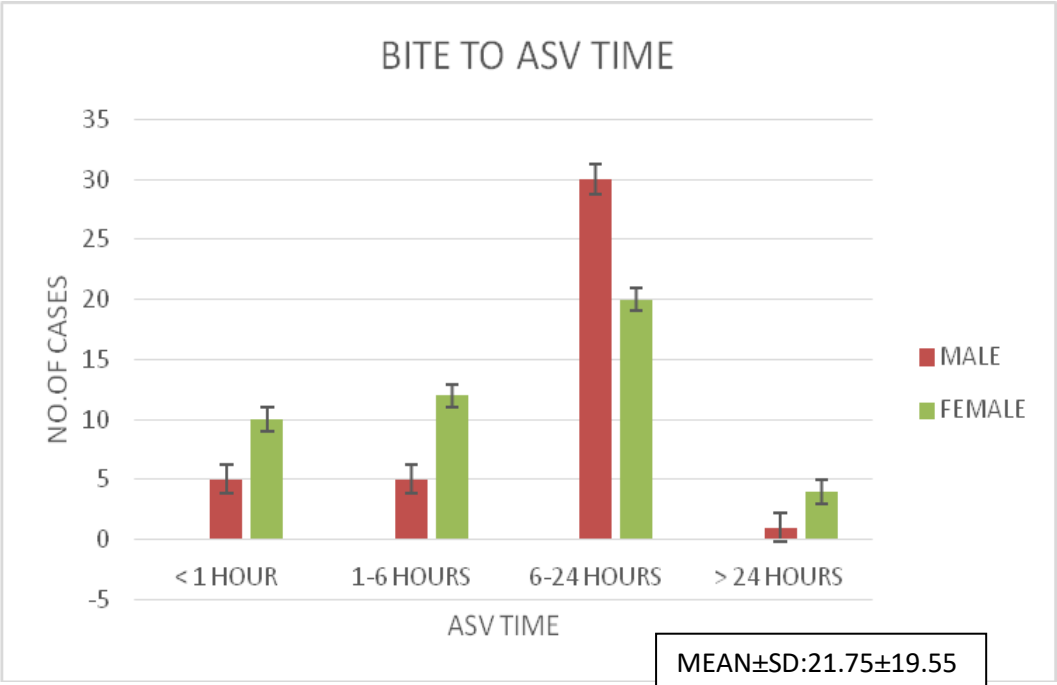
		No.of cases		
ASV	Male	Female	Total (n)	Percentage (%)
Received	41	46	87	81
Not received	8	12	20	19



MEAN±SD: 53.5±47.37

12. BITE TO ASV TIME

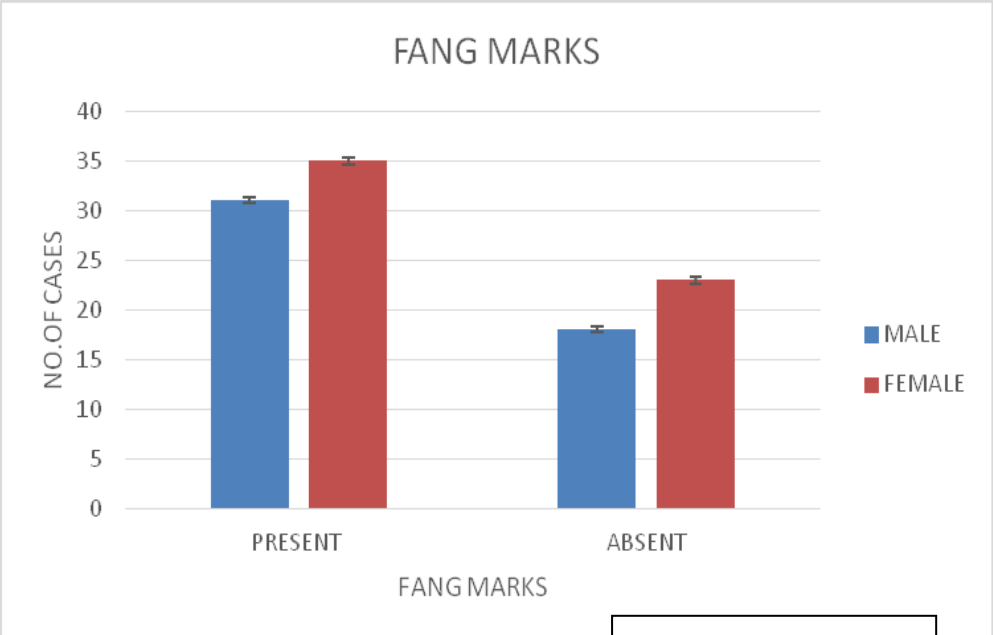
	No.of cases			
Bite to ASV time	Male	Female	Total (n)	Percentage (%)
< 1 Hour	5	10	15	20
1-6 Hours	5	12	17	21
6-24 Hours	30	20	50	54
> 24 Hours	1	4	5	5



MEAN±SD:21.75±19.55

13. FANG MARKS

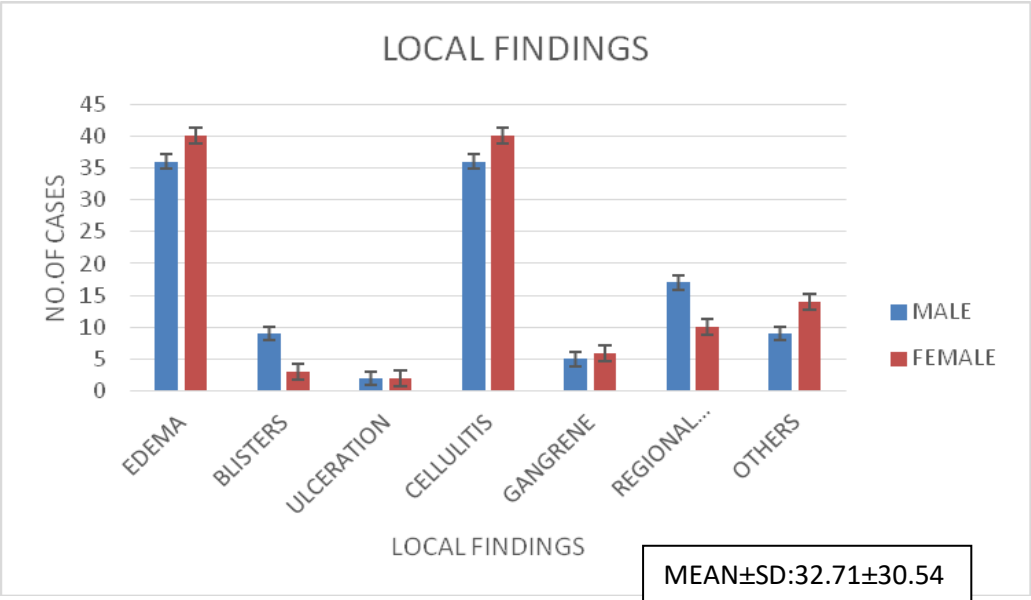
Fang marks	Present	Absent
Male	31	18
Female	35	23
Total (n)	66	41
Percent (%)	62	38



MEAN±SD: 53.5±17.67

14. LOCAL FINDINGS

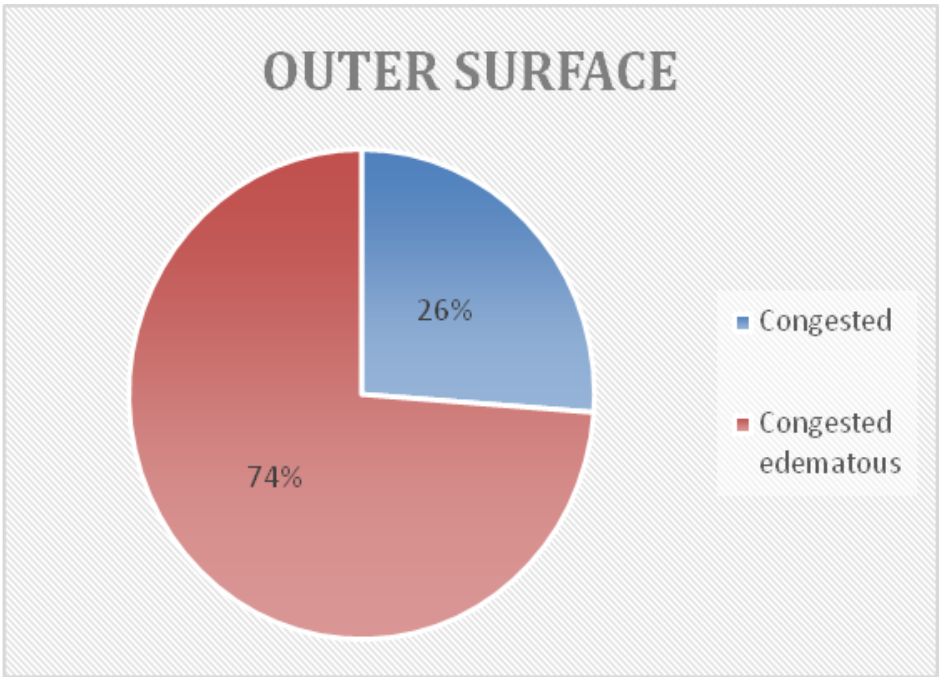
	No.of cases			
Local finding	Male	Female	Total (n)	Percentage (%)
Edema	36	40	76	43
Blisters	9	3	12	7
Ulceration	2	2	4	2
Cellulitis	36	40	76	43
Gangrene	5	6	11	6
Regional lymphadenopathy	17	10	27	15
Others	9	14	23	13

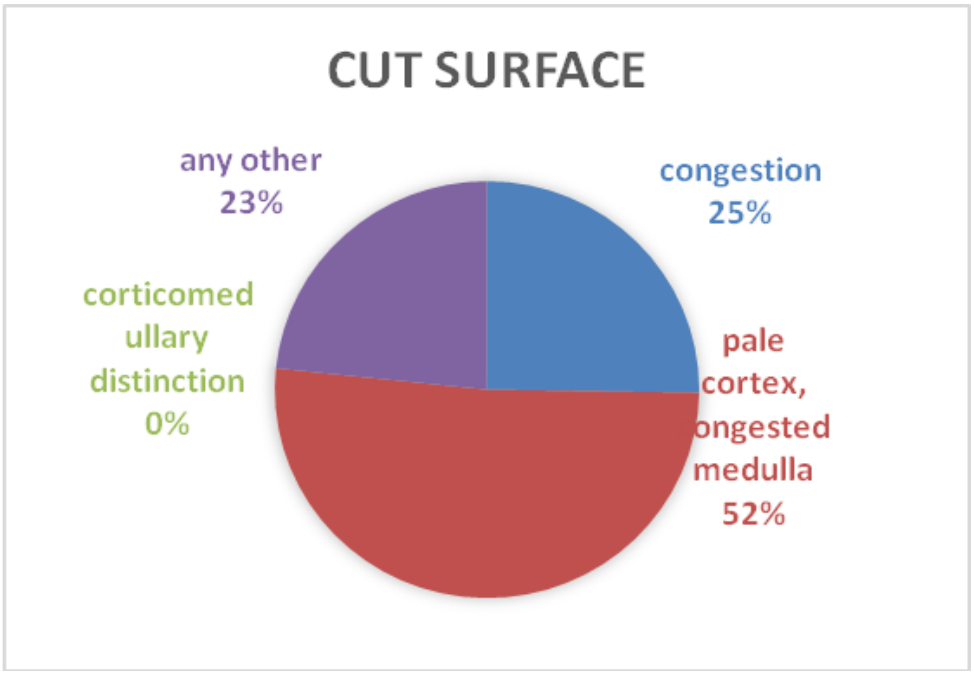


MEAN±SD:32.71±30.54

15. GROSS FINDINGS IN KIDNEY

Outer surface	No.of cases	percentage (%)
Swollen and edematous	79	26
congested	28	74
petechial haemorrhages	0	0
scars	0	0
total	107	
cut surface	no.of cases	percentage (%)
congestion	27	25
pale cortex, congested medulla	55	52
corticomedullary distinction	0	0
any other	25	23
total	107	


OUTER SURFACE
26%
74%
Congested
Congested edematous

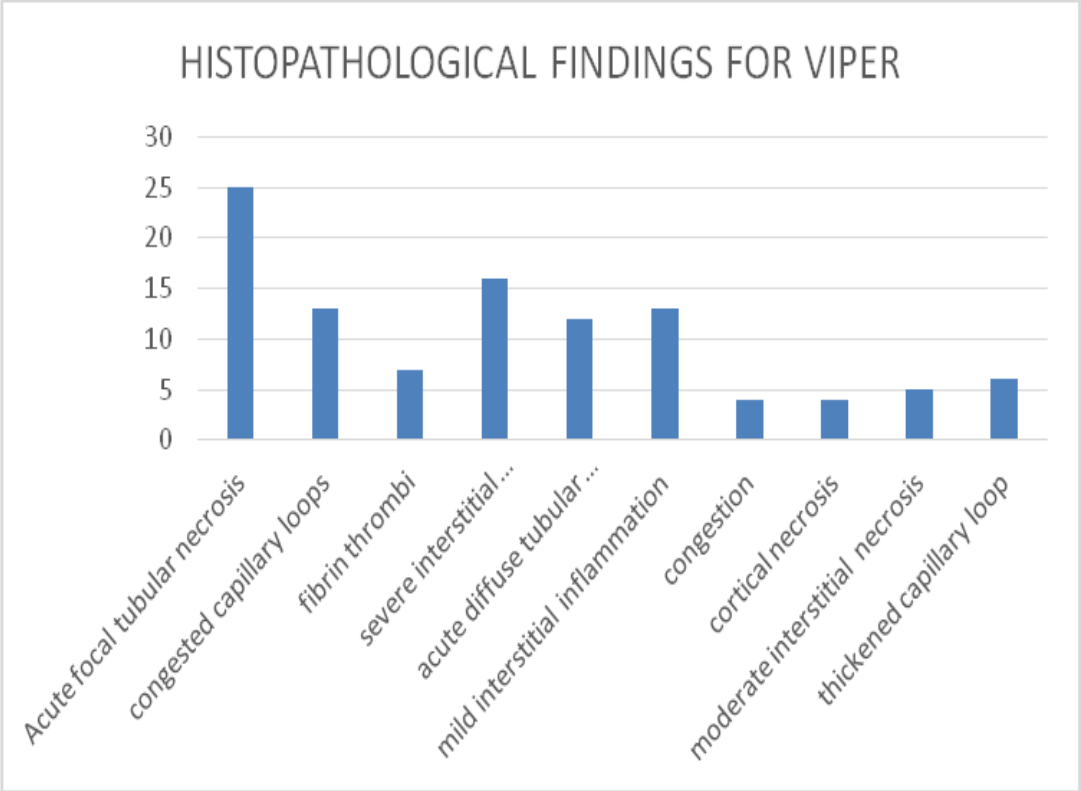

CUT SURFACE
any other 23%
congestion 25%
corticomedullary distinction 0%
pale cortex, congested medulla 52%

16. HISTOPATHOLOGICAL FINDINGS IN KIDNEY

Glomeruli	No.of cases
Normal	0
congested capillary loops	30
DPGN	0
mesangiolysis	0
MPGN	0
thickened capillary loops	10
inflammatory cells	4
cresents	0
fibrin thrombi	10
renal infarction and cortical necrosis tubules	8
normal	0
diffuse acute tubular necrosis	27
focal acute tubular necrosis	41
tubular casts	0
interstitium	
normal	0
congestion	6
inflammation	0
mild	20
moderate	15
severe	30
blood vessels	
normal	0
fibrinoid necrosis	0
thrombiphlebitis	0
other ancillary findings(IHC/IF)	3

HISTOPATHOLOGICAL FINDINGS IN VIPER BITE CASES

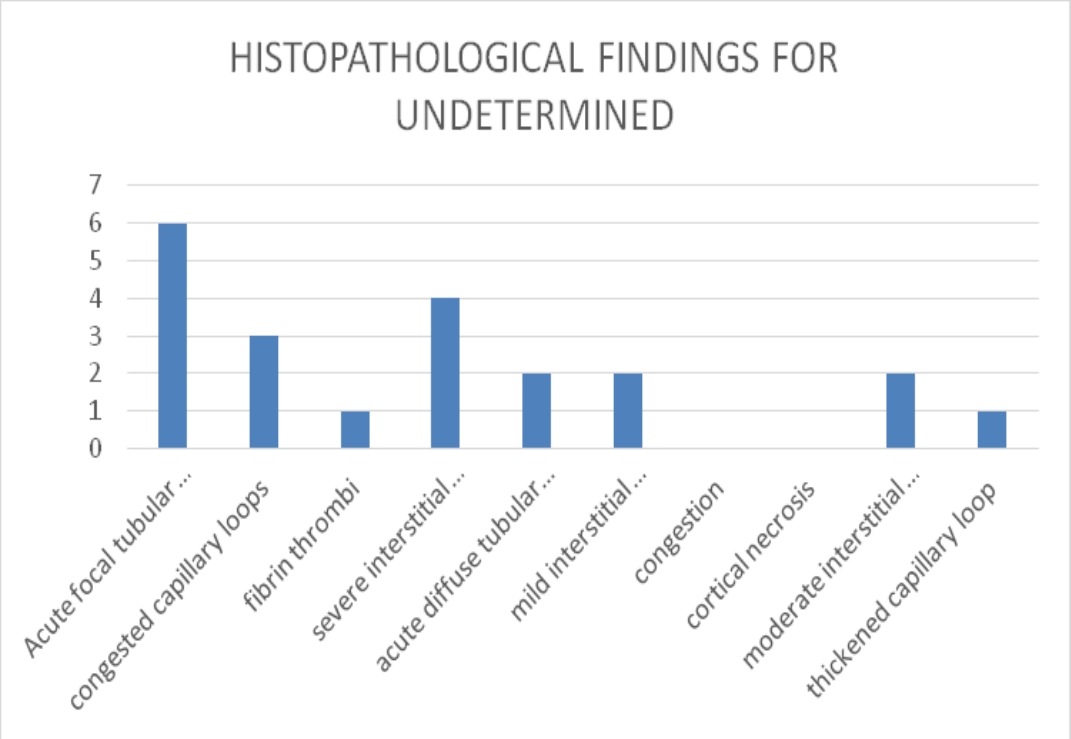
HISTOPATHOLOGICAL FINDINGS	No.of cases	percentage %)
Acute focal tubular necrosis	25	24
Congested capillary loops	13	12
Fibrin thrombi	7	7
Severe interstitial inflammation	16	15
Acute diffuse tubular necrosis	12	11
Mild interstitial inflammation	13	12
Congestion	4	4
Cortical necrosis	4	4
Moderate interstitial necrosis	5	5
Thickened capillary loop	6	6



MEAN±SD:10.5±6.68

HISTOPATHOLOGICAL FINDINGS IN UNDETERMINED CASES

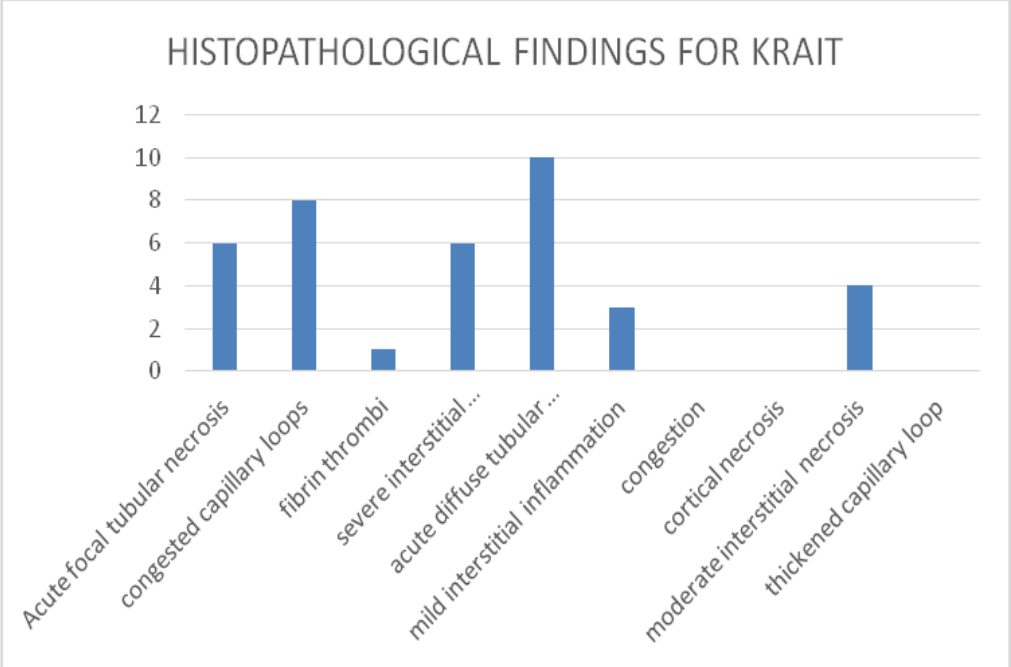
HISTOPATHOLOGICAL FINDINGS	No.of cases	percentage %)
Acute focal tubular necrosis	6	29
Congested capillary loops	3	14
Fibrin thrombi	1	5
Severe interstitial inflammation	4	19
Acute diffuse tubular necrosis	2	9
Mild interstitial inflammation	2	9
Congestion	0	0
Cortical necrosis	0	0
Moderate interstitial necrosis	2	10
Thickened capillary loop	1	5



MEAN±SD:2.1±1.85

HISTOPATHOLOGICAL FINDINGS IN KRAIT BITE CASES

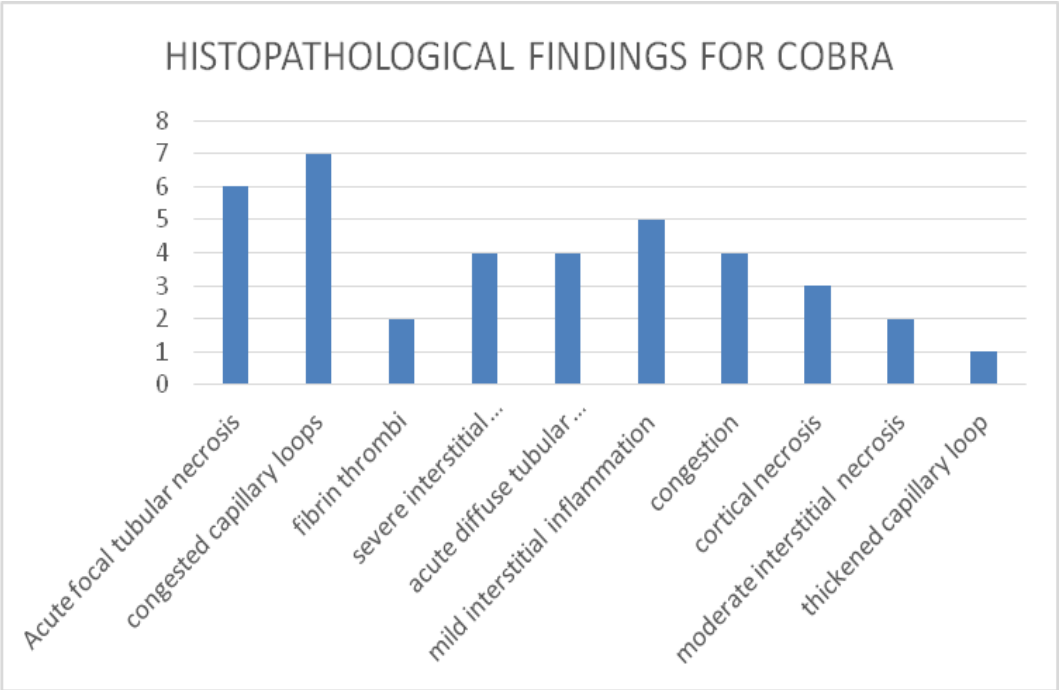
HISTOPATHOLOGICAL FINDINGS	No.of cases	percentage %)
Acute focal tubular necrosis	6	16
Congested capillary loops	8	21
Fibrin thrombi	1	3
Severe interstitial inflammation	6	16
Acute diffuse tubular necrosis	10	26
Mild interstitial inflammation	3	8
Congestion	0	0
Cortical necrosis	0	0
Moderate interstitial necrosis	4	10
Thickened capillary loop	0	0



MEAN±SD:3.8±3.614

HISTOPATHOLOGICAL FINDINGS IN COBRA BITE CASES

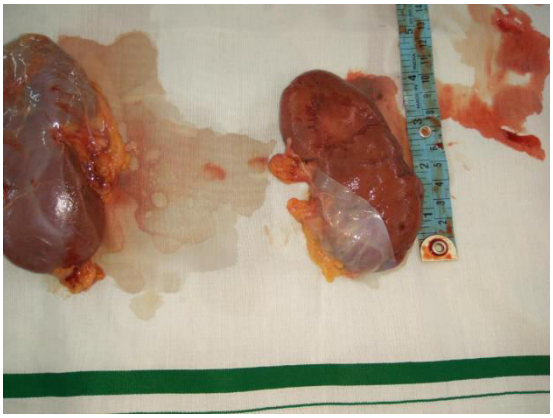
HISTOPATHOLOGICAL FINDINGS	No.of cases	percentage %)
Acute focal tubular necrosis	6	16
Congested capillary loops	7	18
Fibrin thrombi	2	5
Severe interstitial inflammation	4	10
Acute diffuse tubular necrosis	4	11
Mild interstitial inflammation	5	13
Congestion	4	11
Cortical necrosis	3	8
Moderate interstitial necrosis	2	5
Thickened capillary loop	1	3



MEAN±SD:3.8±1.87

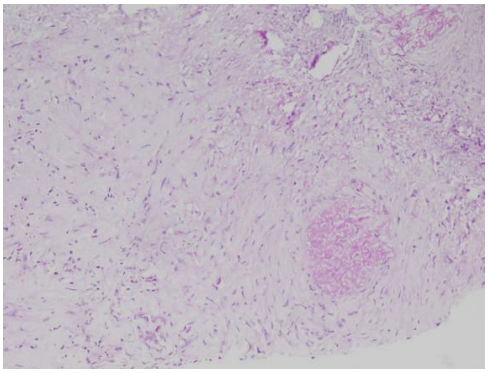
PHOTOGRAPHS

GROSS AND CUTSECTION OF KIDNEY IN SNAKE BITE

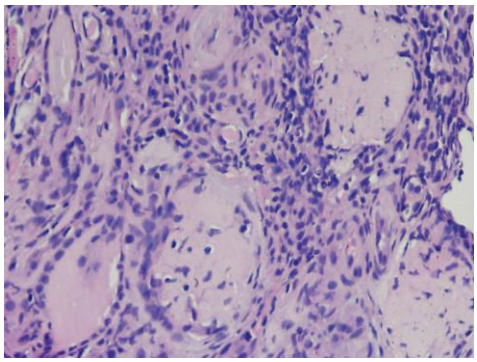


HISTOPATHOLOGICAL PICTURE OF KIDNEY IN SNAKE BITE CASES

ACUTE CORTICAL NECROSIS

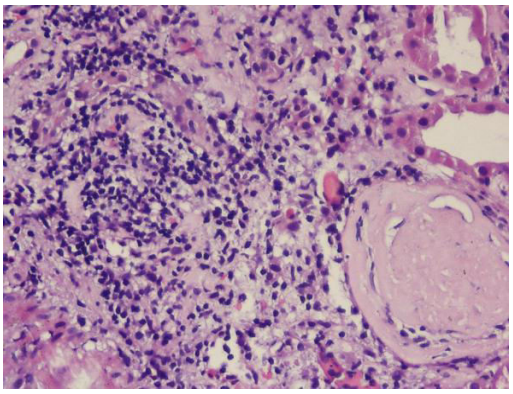


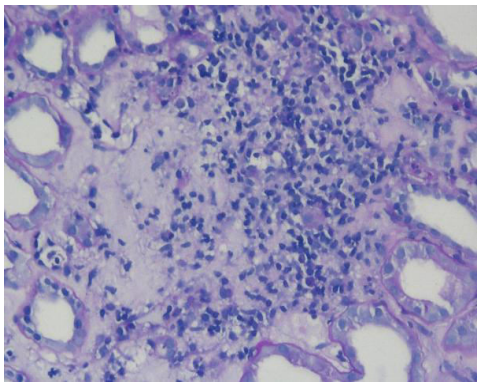
GLOBAL GLOMERULAR SCLEROSIS



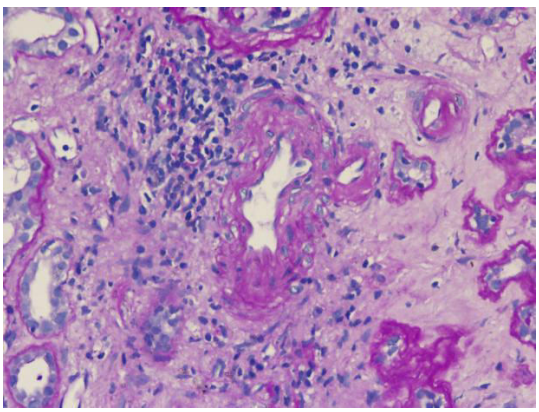
INTERSTITIAL INFLAMMATION (MODERATE)

AND GLOBALLY SCLEROSED GLOMERULUS

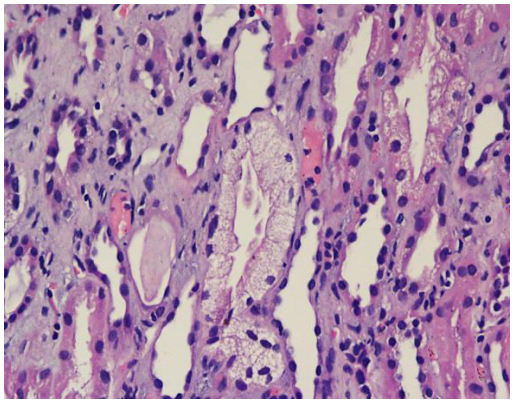




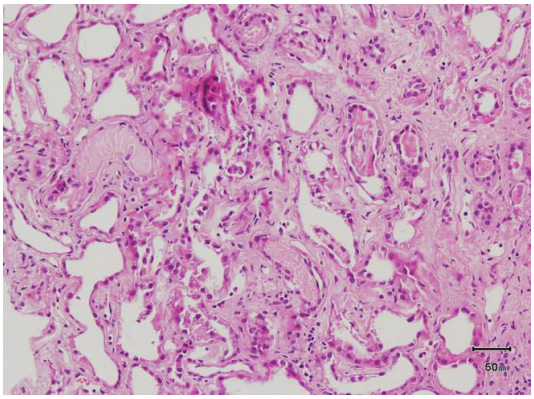
INTERSTITIAL FIBROSIS AND MILD INTERSTITIAL INFLAMMATION



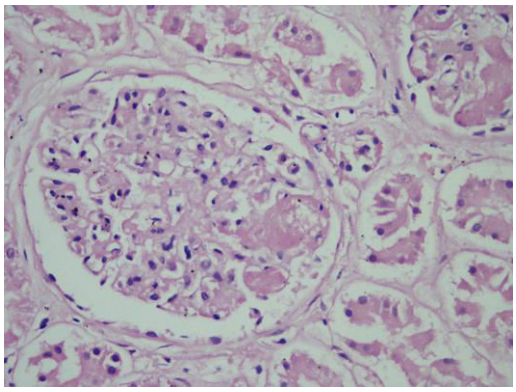
FOCAL ACUTE TUBULAR NECROSIS



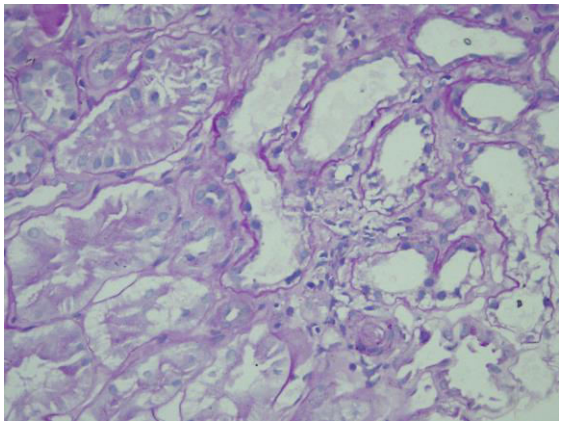
DIFFUSE ACUTE TUBULAR NECROSIS



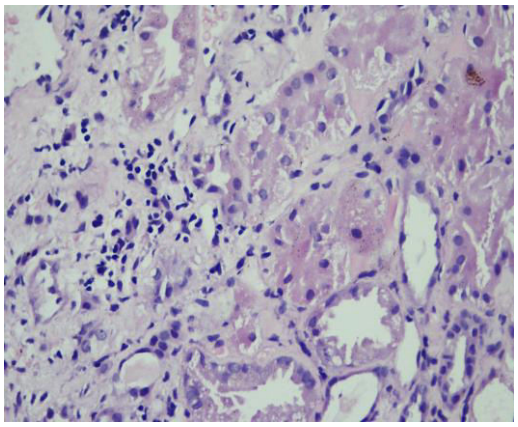
FIBRINOID THROMBI IN GLOMERULI



FIBRIN THROMBI IN GLOMERULI



MILD INTERSTITIAL INFLAMMATION



6. DISCUSSION

1. Age and sex distribution of deceased:

Age group most commonly involved in the study is 50 years and above (n=39,36%). this can be co-related to the fact that elderly people are commonly employed in Madurai for farming and related activities where threat of snake bite is more. Since many rural areas in and around Madurai depend on agriculture as major occupation, the age group affected is skewed to the elderly spectrum. This study is in concordance with Venkatesan M, Dongre AR et.al[1] study, which was conducted in rural district of TamilNadu and owing to increased susceptibility, deaths are common in this age group. This percentile differs from other studies such as Yogesh C et.al, Bhardwaj A et.al and Chugh et,al[2] where mean age of subjects was 33±10 years. In our study percentage of males affected 45% (n=49) and percentage of females affected is 54.3% (n=58). This is in contrast with previous studies such as Liw et.al [3], J.S.Whitehall et.al [4], Bhalla et.al [6] and Alakesh Halder et.al [5] in which male preponderance was noted. More number of working women in Madurai district can be a reason for such discordance.

2. Occupation pattern of deceased:

Agriculture labourer (n=63, 59%) is the most affected group followed by non-agriculture labourer (n=19, 18%). Adult farmers are chief victims of snake bite because of their active involvement in field works. Most of the farm workers do not have proper footwear for protection against this problem.

Bhalla G et.al [6], study pointed out to a similar proportion of farmers involved. Bhat et.al [7], Saini et.al [8] and Sarangi et.al [9] showed the incidence in farmers to be 75%, 72% and 78% respectively.

3. Education of deceased:

Uneducated members of society (n=83, 76%) formed the major portion of affected ones. Harshvardhan C et.al [10] also noticed a similar picture.

4. Type of snake:

Most of the species of snake bite in our study are Viper (n=49; 46%) followed by neurotoxic snake bite (Krait n=24; 22% & Cobra n=21; 20%). This is consistent with Harshavardan et.al study[10], in which vasculotoxic bite was predominant Whithall et al[5] were also consistent with current study.

5. Location at the time of bite:

In our study, bites were encountered in outdoor area more (n=79, 74%) than indoor area (n=28, 26%). Predominant cases were reported from agricultural fields and during activity near the house premises. Bite near the house premises are due to the rural environment with huts and houses, dry cowdung, dry firewood, firm tools being kept encourages rats, mice and lizards which are the prey for snakes. This percentage of outdoor predominance is in accordance with Vanamali et.al [(11)].

6. Time of bite:

Time period during which snake bite was most recorded is 6pm-12am (n=40, 37%) followed by 6am-12pm (n=38, 36%). This is in line with previous studies such as Bhalla et.al [(6)] and Alakesh Halden et.al [(5)]. In our study snake bite commonly occured in the dark hours of the day.

Since snakes are nocturnal in nature, diurnal variation is observed in our study. Similar, diurnal variation was also observed by Bawaskar HS et.al [(12)] and Montaro NP et.al [(13)].

7. Seasonal variation:

Maximum fatally occurred during the winter (n=51, 48%) and rainy seasons (n=34, 32%). Most bites observed are during the months of July - November which represent the monsoon. Snakes are known to

be more active this season. This poses increased risk to farmers as they venture out to the fields to harvest their crops. Other studies such as Brunda G et.al [14] and Kularatne SAM [15] also noted a similar increase in incidence of snake bites in monsoon when compared to summer.

8. Activity at the time of bite:

Snake bite during activity (n=92, 86%) predominated the snake bite during sleep (n=15, 14%). This is in contrast with Rupinder sharma et.al [16] who demonstrated bite during sleep was more compared to bite during activity. The difference in numbers can be explained by the fact that neurotoxic bites especially Krait is most common during night and at indoors and it is predominant in North India.

9. Site of bite:

Lower limb was the commonest site of snake bite in our study followed by upper limb and abdomen. This is similar to findings of most of studies such as Yogesh C et.al [17], KulKarni et.al [18] and Harshavardhan et.al [10] and Alakesh Halden et.al [5]. People accidentally step onto the snake during walking. This snake bite is more common in the lower limb.

10. First aid prior to ASV:

In most number of cases in our study, no first aid was given before ASV (n=92, 86%), torniquet was inserted in 7% (n=7) of cases and herbal medicines was applied in 7% (n=8) of cases. Nonmedical treatment was not taken in a majority of cases. This is in discordance with Singh RR et.al in which 43% took non medical treatment before ASV. Though most of our study group are uneducated, sensitisations among people even in rural areas has prevented them from taking non medical measures.

11. ASV received or not:

Majority of snake bite cases in our study received ASV (n=87, 81%). Those cases that did not receive ASV were either fatal and died within hours of reporting to hospital or due to very late reporting.

12. Bite to ASV time (Bite to needle time):

Bite to ASV time is more than 6 hours (late SAV) in 66.8% (n=63) cases and less than 6 hours (early SAV) in 32.2 % (n=44) cases. Bite to needle time is higher in our study due to the delay in reporting of patients. This is in concordance with Sharma N et.al [(20)] and Bebarta V et.al [(21)] and is discordance with Suchithra et.al [(22)].

13. Fang marks:

Fang marks were present in 62% (n=66) of cases and absent in 38% (n=41) of cases. This is in line with Chidananda PS Rao et.al [23] who observed a similar ratio in their study.

14. Local findings:

Local pain and swelling is present in 43% (n=76) cases. Rubina Naqir et.al [25] had a similar findings. But patel TB et.al [24] and Suchitra N et.al [22] reported 95% of cases with local pain and swelling. This was partly due to late presentation of patients in our study leading to settlement of local symptoms. Cellulitis was also present in majority of cases in our study (n=76, 43%) Harshavardhan et.al [10] reported that 96% of cases had cellulitis.

12. Gross findings of kidney:

In our study, major gross findings noted was swelling and edema (n=79, 74%) followed by congestion (n=28, 26%) on outer surface. On cut section, medullary congestion was noted in 52% cases (n=55) and Petechial haemorrhages in corticomedullary junction noted in 23% (n=25) cases. This is in unison with Yogesh C et.al [17] who observed congestion in majority of cases. Findings of Petechial haemorrhage is also consistent with Alakesh Halden et al [5] in which 21% cases

reported Petechial haemorrhage. Pale cortex and congested medulla is seen in 55 cases (n=52%) in our study. Such a finding has been explained of ischaemic origin by Duff & Murray [27] and Lauler & Schreiner [26].

13. Histopathological findings of kidney:

Among the Viper cases reported (n=49, 46%). Glomerular changes noted in 26 cases 53% (congested capillary loops in 13 cases, thickened capillary loop in 6 cases and fibrin thrombi in 7 cases).

Cortical necrosis in 4 cases(4%).

Tubular changes in 37 cases (76%) (acute diffuse tubular necrosis in 12 cases, acute focal tubular necrosis in 25 cases).

Interstitial changes in 34 cases (69%) (mild interstitial inflammation in 13 cases, moderate interstitial inflammation is 5 cases, severe interstitial inflammation in 16 cases).

Increased prevalence of ATN in Viper bite in our study is in consistent with M Pal.et.al [28] and Alakesh Halden et.al [5]. upto 70% of the protein content of Viper venom is phospholipase A_2, present in the form of atleast seven isoenzymes. It causes hemolysis,rhabdomyolysis, presynaptic neurotoxicity, vasodilation & shock and interaction with monoamine receptors. There is evidence that renal blood flow

diminishes substantially in toxic acute tubular necrosis, apparently as a result of arteriolar vasocontriction. Many investigators believe that tubuar obstructive casts play a major role in the pathogenesis of ARF. This obstruction would increase intratubular pressure, contributing to the backleak of tubular epithelium. This tubular backleak causes interstitial edema which further impairs renal function.

The renal lesion that carries the most sinister prognosis is acute cortical necrosis. Snake bite is the second most common cause of acute cortical necrosis in India, comprising 4% of Viperine bites in our study. Autopsy specimens showing diffuse cortical necrosis disclosed a narrow subcapsular zone containing a few tubules that appeared normal. Broad Ischaemic area was noted in the cortex. Fibrin thrombi of 7% is in unison with Chug et.al [(2)] study. The Ischaemic zone was found seperated from the medulla by a zone of tissue that appeared hyperemic and contained a dense leukocytic infiltrate. The histologic changes varied with duration of illness. Glomerular changes in snake bite can be attributed to the vasculotoxic action of venom or hypersensistivity to the venom or immune complex disease. Venom induced platelet release reaction initiates mesangiolysis.

In mesangiolysis, visceral epithelial cells showed intracellular edema and mesangial hypercellularity were found at a late stage in the evolution of glomerular change.

Presence of fibrin thrombi in the renal microvasculative and in the glomerular caapillaries and microangiopathy in acute cortical necrosis strongly suggests that DIC plays a major pathogenetic role in the lesions of snake bite induced acute cortical necrosis. Experimentally, acute bilateral cortical necrosis can be produced by inducing a Shwartzman phenomenon with endotoxin. The endotoxin infusion model has relevance to the pathogenesis of renal lesions in snake bite.

Among the krait cases reported, (n=24, 22%) histopathological changes noted in kidney are as follows:

Glomerular changes in 9 cases (congested capillary loops in 8 cases & fibrin thrombi in 1 case).

Tubular changes in 16 cases (focal ATN in 6 cases and diffuse ATN in 10 cases) and interstitial changes in 12 cases.

This non-neurotoxic profile of krait envenomation was experimentally dealt in Monhkon Charoen pita K chai et.al [(29)] . krait venom induced skeletal muscle damage is characterised by

hypercontraction of myofilaments, diruption of plasma membrane and tissue necrosis including release of CK.

Renal damage can be induced by direct and indirect myotoxic effects of toxins. The indirect effects cause nephrons to be overloaded by degraded proteins, including myoglobin from decayed tissue tubules, which result in secondary acute kidney injury; whereas direct effects cause damage to the kidney cells due to cytotoxicity [30,31]. however the myotoxins in the Krait venom is still not characterised.

Glomerular and vessels congestion, shrinking of glomeruli, hemorrhage & necrosis of proximal tubules were observed in Krait envenomation by Al-Mamun et.al[32].

Among the cases of Cobra bite reported in our study (n=21 cases).

Glomerular lesions noted in 10 cases (congested capillary loops in 7 cases, fibrin thrombi in 2 cases and thickened capillary loop in 1 case).

Tubular lesions noted in 10 cases (acute focal necrosis in 6 cases and diffuse type in 4 cases).

Interstitial changes noted in 11 cases and renal cortical necrosis noted in 3 cases.

Our study is in concordance with Mukhopadhyay et.al [33] who demonstrated changes of tubular necrosis, cortical necrosis and interstitial nephritis in bites due to Indian Cobra (Naja Naja). our study varies with previous documented studies such as Chugh K.S et.al [2], Mittal. B.V et.al [34] and Ali G et.al [35]. where hemotoxic bites were only reported to cause nephropathy. The study highlights the renal involvement in Cobra bite cases contrary to the fact that they are mainly neurotoxic. Two possible mechanisms can be postulated:

1. Mixed toxins with intrinsic activity on the kidneys.

2. Immunologically mediated nephropathy.

Among the envenomation of snakes of undetermined species in our study (n=13 cases).

Tubular lesions noted in 8 cases (6 cases of focal ATN and 2 cases of diffuse ATN).

Glomerular lesions noted in 5 cases (congested capillary loop in 3 cases, fibrin thrombi in 1 case, thickened capillary loop in 1 case) and Interstitial inflammation noted in 8 cases.

7. CONCLUSION

In the present study, out of 107 cases of snake bite, age group commonly involved is 50 years and above, most affected being agricultural labourer, maximum fatality occurred at winter and during activity. Lower limb is the commonest site involved and majority of cases received antisnake venom, eventhough bite to ASV time is more. Among the local findings, local pain and swelling predominated. Histopathological study of kidney revealed that tubular lesions (acute focal tubular necrosis) predominant in Viperine bites, tubular (diffuse ATN) and interstitial changes (severe inflammation) in Krait bite and glomerular (congested capillary loop) and interstitial (severe inflammation) changes in Cobra bite.

Prevalence of acute kidney injury in our study is 79.5% which is very high compared to previous studies. Further monitoring of renal parameters even in neurotoxic snake bite and prompt dialysis should be done). This indicates the need for further large scale investigations with detailed pathology of neurotoxic snake bite with ultrastrctural and molecular studies.

Advanced age and increased time interval from snake bite to antivenom administration were independent risk factor for snake bite induced AKI. Special investigation into the biochemical and immunologic components of Cobra and Krait venom should also be considered. Rationalisation of antisnake venom therapy be done to prevent ASV related kidney injury.

Instead of the single polyvalent antisnake venom, feasibility of conducting research into the use of bivalent antivenoms should be considered as a AKI due to antivenoms can be reduced to the minimum. This will greatly increase the efficacy and minimize side effects, so that specific antivenom based on clinical picture and investigations can be given.

8. ANNEXURES

A. BIBLOGRAPHY

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B. PROFORMA

SECTION-A

1. Post-mortem number:

2. Age:

3. Sex: Male / Female

4. Occupation:

a. Agriculture (labourer)

b. Non Agriculture (labourer)

c. Others (specify)

5. Education:

a. Primary school

b. Higher secondary

c. Graduate

d. Uneducated

6. Type of snake:

a. Viper

b. Cobra

c. Krait

d. Unidentified

7. Location at the time of bite:

a. Indoor	
b. Outdoor i. Agriculture fields	
ii. House premises	
iii. Others	

8. Time of bite:

a. 6AM to 12PM

b. 12PM to 6PM

c. 6PM to 12AM

d. 12AM to 6AM

e. Exact time not known

9. Seasonal Variation:

a. Summer (March-June)

b. Rainy (July-October)

c. Winter (November-February)

10.Activity at the time of bite:

a. Sleeping

b. Doing household activities

c. Work in agricultural fields

d. Others

11. Site of bite:

a. Lower limb

b. Upper limb

c. Head,body and neck

12. Any precautions taken:

13. First Aid prior to Anti Snake Venom:

a. No first aid

b. Tourniquet

c. Incision

d. Application of herbal medicine

e. Suction of venom from the bite site

14. Received ASV or not: Yes / No If Yes, how many vials:

15. Bite to Anti Snake Venom time (Time delay to receive Anti Snake Venom):

a. <1 hour

b. 1-6 hours

c. 6-24 hours

d. >24 hours

16. Duration of survival:

SECTION-B (On Autopsy)

1. Site of bite:
2. Fang Marks: Present / Absent If present: Single/Multiple
3. Local findings:

a. Edema

b. Blisters

c. Ulceration

d. Cellulitis

e. Gangrene

f. Regional lymphadenopathy

g. Others

4. Gross findings on internal organs

a. Kidney:

Size : Right kidney

Left Kidney

	Left Kidney	Right kidney
Outer surface:		
Swollen and edematous		
Congested		
Petechial hemorraghes		

Cut surface:

Congestion

Pale cortex, congested medulla

Cotricomedullary distinction

Any other

SECTION-C

Histopathological Findings

1.Kidney:

Glomeruli	normal, Congested capillary loops, DPGN, Mesangiolysis, MPGN, Thickened Capillary loops, Inflammatory cells, crescents, Fibrin thrombi
Renal infarction and Cortical necrosis	
Tubules	Normal/ Acute tubular necrosis (Diffuse/focal), tubular casts, tubular epithelial cells
Interstitium	Normal/ congestion/ inflammation mild/moderate/severe
Blood vessels	Normal/ fibrinoid necrosis/ thrombophlebitis
Other changes	Additional with diabetes/ hypertension to be noted.

Other ancillary findings (IHC/ IF)

C. MASTER CHART

Sl. No.	PM No	Age	Sex	Occupation	Education	Type of Snake	Location at the time of Bite	Time of Bite	Seasonal Variation	Activity At the time of bite	Site of bite	First Aid prior to ASV	Received ASV or Not	Bite to ASV Time	Duration of survival	Fang marks	Local findings	Gross findings On Kidney			HPE Findings on Kidney	Other Findings
																		Size	Outer Surface	Cut surface		
1.	2682/16	64	F	Agriculture labourer	Uneducated	Unidentified	Agriculture Field	12.PM	Rainy	Work	Lower Limb	-			1Day	Absent	-		Congested	Petechial haemorhage	Acute (focal) Tubular Necrosis	Subarachnoid Haemorrhage Intraventricular haemorrhage
2.	2737/16	45	M	Non agriculture labourer	Uneducated	Unidentified	Outdoor House premises	7pm	Rainy	others	Lower Limb	-			9Days	Present	Edema, blisters		Congested	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
3.	2829/16	16	M	Student	Higher secondary	Viper	Others	6pm-12am	Rainy	Others	Lower Limb	-	35 Vials	7 hours	5 Days	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Disseminated Intra vascular Coagulation, Pulmonary Edema
4.	2841/16	45	F	Non agriculture labourer	Uneducated	Viper	Others	6pm-12am	Rainy	Others	Lower Limb	-			3 Days`	Present	Edema, Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
5.	3004/16	40	F	Others	Uneducated	Viper	Others	6am-12pm	Rainy	Others	Lower Limb	-			3 hours	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Pulmonary edema
6.	3100/16	60	M	Non agriculture labourer	Uneducated	Viper	Outdoor House premises	6am-12pm	Rainy	Household activities	abdomen	-			8days	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Fibrin thrombi glomerulus Focal acute tubular Necrosis Mild Interstitial inflammation	Pulmonary edema
7.	3136/16																					
8.	3248/16	46	F	Agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Rainy	Household Activities	Lower Limb		13 Vials	6 hours	5 days	Present	Ulceration Cellulitis Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Sepsis, increased CPK Increased LDH
9.	3370/16	40	F	Agriculture labourer	Uneducated	Krait	Indoor	6pm to 12am	Rainy	Household Activities	Lower Limb				9 days	Absent	Cellulitis Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	Metabolic Encephalopathy, Sepsis.
10.	3378/16	53	M	agriculture labourer	Uneducated	Krait	Others	6 am to 12pm,	Winter	Agriculture Work	Lower Limb				8 days	Absent	Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
11.	47/17	25	F	agriculture labourer	Primary School	Unidentified	Agriculture Field	12pm to 6pm	Winter	Agriculture Work	Lower Limb				5 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial Inflammation	

12.	59/17	49	F	agriculture labourer	Uneducated	Viper	Agriculture Field	6pm to 12pm	Winter	Agriculture Work	Upper Limb				1 day	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
13.	207/17	23	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				11 days	Absent	Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
14.	371/17	51	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				1 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	Intra cerebral haemorrhage
15.	518/17	62	F	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				14 days	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
16.	551/17	65	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				1 day	Present	Nil		Congested Edematous	Petechial Haemorrhage	Congested Capillary Loops in glomeruli, interstitial congestion, mild interstital inflammation, Chronic pyelo nephritis	
17.	622/17	60	F	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				2 hours	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis Severe Interstitial Inflammation, Congested Capillary Loops in glomeruli,	Intra cerebral haemorrhage
18.	786/17	8	F	Student	Primary School	Unidentified	Out door	6pm to 12am	Summer	Others	Lower Limb				1 day	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Pulmonary Edema
19.	928/17	60	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6pm to 12am	Summer	Agriculture Work	Lower Limb				7 days	Absent	Nil		Nil	Nil	Nil	Subdural, Subarachnoid Haemorrhage Left hemo thorax

20.	1008/17	38	F	agriculture labourer	Uneducated	Unidentified	Indoor	6am to 12pm	Summer	Others	Lower Limb				3 days	Present	Congestion & Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
21.	1167/17	40	F	agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Summer	Others	Lower Limb				3 days	Present	Congestion & Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis Congested Capillary Loops	Pulmonary Edema
22.	1170/17	48	F	agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Summer	Others	Lower Limb				1 day	Present	Congestion & Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis mild interstital inflammation	
23.	1200/17	51	M	agriculture labourer	Uneducated	Krait	Agriculture Field	12pm to 6pm	Summer	Agriculture Work	Lower Limb				7 days	Present	Edema, Ulceration		Congested Edematous	Petechial Haemorrhage	Inflammation In glomeruli, Acute (focal) Tubular Necrosis	Pulmonary Edema
24.	1368/17	45	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6pm to 12am	Summer	Agriculture Work	Lower Limb				18 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Inflammation In glomeruli,	
25.	1403/17	46	F	agriculture labourer	Uneducated	Unidentified	Indoor	12am to 6am	Summer	Sleeping	Lower Limb				17 days	Absent	Edema Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
26.	1508/17	50	F	agriculture labourer	Uneducated	Cobra	Agriculture Field	6am to 12pm	Summer	Agriculture Work	Lower Limb				3 days	Present	Edema & Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis Cortical Necrosis	
27.	1661/17	65	F	agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Summer	Sleeping	upper Limb				4 days	Absent	Edema & Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis mild interstital inflammation	
28.	1712/17	11	F	Student	Primary School	Unidentified	Indoor	6pm to 12am	Summer	Others	Lower Limb				5 days	Present	Edema				Acute (focal) Tubular Necrosis Cortical Necrosis	
29.	1808/17	55	F	agriculture labourer	Uneducated	Unidentified	House Premises	6pm to 12am	Rainy	Household Work	Lower Limb				4 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Severe interstital inflammation	
30.	2015/17	55	F	agriculture labourer	Uneducated	Unidentified	Agriculture Filed	6pm to 12am	Rainy	Agriculture Work	Lower Limb				3 days	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Sepsis, retro peritoneal haematoma
31.	2022/17	55	F	agriculture labourer	Uneducated	Unidentified	Houses Premises	12pm to 6pm	Rainy	Others	Lower Limb				18 days	Absent	--		--	--	--	Left occipital lobe infarct
32.	2155/17	48	M	agriculture labourer	Uneducated	Unidentified	Indoor	6am to 12pm	Rainy	Sleeping	Upper Limb				6 Hours	Present	Edema		--	--	--	

33.	2184/17	45	M	Non agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Rainy	Others	Upper Limb				12 Hours	Present	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Hypoxic Ischaemic Encephalopathy, respiratory failure
34.	2191/17	56	M	Non agriculture labourer	Uneducated	Unidentified	Others	6pm to 12am	Winter	Others	Lower Limb				5 days	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage		
35.	2272/17																					
36.	2281/17																					
37.	2323/17	60	M	agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Rainy	Sleeping	Lower Limb				5 days	Present	Congestion Edema Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Congested Capillary loops	
38.	2335/17	16	F	Student	Higher Secondary	Cobra	Indoor	12am to 6am	Rainy	Sleeping	Upper Limb		12 vials	6 hours	3 days	Absent	--		--	--	Nil	Respiratory failure
39.	2468/17	41	M	agriculture labourer	Uneducated	Krait	Indoor	6pm to` 12am	Rainy	House hold Work	Lower Limb				3 days	Present	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	Perinephric haematoma
40.	2741/17	60	F	Non agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Rainy	Others	Lower Limb				19 days	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	Metabolic encephalopathy
41.	2767/17	52	M	Non agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Rainy	Others	Lower Limb				1 day	Present	Congestion Edema				Acute (diffuse) Tubular Necrosis	Right Hemiparesis
42.	2918/17																					
43.	2970/17																					
44.	3190/17	45	F	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6pm to 12am	Rainy	Agriculture work	Upper Limb				1 day							Neuro Toxic, Respiratory failure
45.	3264/17	55	F	agriculture labourer	Uneducated	Unidentified	Indoor	12am to 6am	Winter	Sleeping	Lower Limb		12 vials	4 hours	6 hours	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	Neuro Toxic, Respiratory failure
46.	3356/17	43	M	Non agriculture labourer	Uneducated	Krait	Indoor	12am to 6am	Winter	Sleeping	Gluteal region				4 days	Present						Neuro Toxic, Respiratory failure
47.	149/18	2	M	Student	Primary School	Cobra	Indoor	6pm to 12am	Winter	Others	Upper Limb				1 day	Present						Neuro Toxic, Disseminated Intra vascular coagulation
48.	242/18	36	F	agriculture labourer	Uneducated	Unidentified	Others	12pm to 6pm	Winter	Others	Lower Limb				6 hours	Present						Neuro Toxic, Respiratory failure

49.	345/18	22	M	agriculture labourer	Uneducated	Unidentified	Others	12pm to 6pm	Winter	Others	Lower Limb				3 days	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	
50.	398/18	45	M	agriculture labourer	Uneducated	Unidentified	Others	12am to 6am	Winter	Others	Lower Limb				5 days	Absent	Congestion Edema					Neuro Toxic, Respiratory failure
51.	401/18	35	F	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				14 days	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
52.	407/18	60	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				7 days	Present	Nil		Congested Edematous	Petechial Haemorrhage	Congested Capillary Loops in glomeruli, interstitial congestion, mild interstital inflammation, Chronic pyelo nephritis	
53.	415/18	42	F	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				6 hours	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis Severe Interstitial Inflammation, Congested Capillary Loops in glomeruli,	Intra cerebral haemorrhage
54.	480/18	8	F	Student	Primary School	Unidentified	Out door	6pm to 12am	Summer	Others	Lower Limb				1 day	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Pulmonary Edema
55.	502/18	38	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6pm to 12am	Summer	Agriculture Work	Lower Limb				9 days	Absent	Nil		Nil	Nil	Nil	Subdural, Subarachnoid Haemorrhage Left hemo thorax
56.	540/18	60	F	agriculture labourer	Uneducated	Unidentified	Indoor	6am to 12pm	Summer	Others	Lower Limb				5 days	Present	Congestion & Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
57.	617/18	40	F	agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Summer	Others	Lower Limb				4 days	Present	Congestion & Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis Congested Capillary Loops	Pulmonary Edema
58.	681/18	48	F	agriculture labourer	Uneducated	Unidentified	Indoor	6pm to	Summer	Others	Lower Limb				1 day	Present	Congestion & Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular	

								12am													Necrosis mild interstital inflammation	
59.	772/18	75	M	agriculture labourer	Uneducated	Krait	Agriculture Field	12pm to 6pm	Summer	Agriculture Work	Lower Limb				8 days	Present	Edema, Ulceration		Congested Edematous	Petechial Haemorrhage	Inflammation In glomeruli, Acute (focal) Tubular Necrosis	Pulmonary Edema
60.	790/18	18	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6pm to 12am	Summer	Agriculture Work	Lower Limb				16 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Inflammation In glomeruli,	
61.	815/18	36	F	agriculture labourer	Uneducated	Unidentified	Indoor	12am to 6am	Summer	Sleeping	Lower Limb				15 days	Absent	Edema Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
62.	890/18	56	F	agriculture labourer	Uneducated	Cobra	Agriculture Field	6am to 12pm	Summer	Agriculture Work	Lower Limb				5 days	Present	Edema & Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis Cortical Necrosis	
63.	931/18	65	F	agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Summer	Sleeping	upper Limb				6 days	Absent	Edema & Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis mild interstital inflammation	
64.	990/18	3	F	Student	Primary School	Unidentified	Indoor	6pm to 12am	Summer	Others	Lower Limb				6 days	Present	Edema				Acute (focal) Tubular Necrosis Cortical Necrosis	
65.	1010/18	25	F	agriculture labourer	Uneducated	Unidentified	House Premises	6pm to 12am	Rainy	Household Work	Lower Limb				3 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Severe interstital inflammation	
66.	1058/18	48	F	agriculture labourer	Uneducated	Unidentified	Agriculture Filed	6pm to 12am	Rainy	Agriculture Work	Lower Limb				3 days	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Sepsis, retro peritoneal haematoma
67.	1090/18	65	F	Agriculture labourer	Uneducated	Unidentified	Agriculture Field	12.PM	Rainy	Work	Lower Limb	-			1 Day	Absent	-		Congested	Petechial haemorhage	Acute (focal) Tubular Necrosis	Subarachnoid Haemorrhage Intraventricular haemorrhage
68.	1105/18	71	M	Non agriculture labourer	Uneducated	Unidentified	Outdoor House premises	7pm	Rainy	others	Lower Limb	-			10 Days	Present	Edema, blisters		Congested	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
69.	1158/18	16	M	Student	Higher secondary	Viper	Others	6pm-12am	Rainy	Others	Lower Limb	-	35 Vials	7 hours	6 Days	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Disseminated Intra vascular Coagulation, Pulmonary Edema
70.	1173/18	45	F	Non agriculture labourer	Uneducated	Viper	Others	6pm-12am	Rainy	Others	Lower Limb	-			3 Days`	Present	Edema, Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
71.	1205/18	40	F	Others	Uneducated	Viper	Others	6am-12pm	Rainy	Others	Lower Limb	-			3 hours	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Pulmonary edema
72.	1237/18	60	M	Non	Uneducated	Viper	Outdoor	6am-	Rainy	Household	abdomen	-			9 days	Present	Edema,		Congested	Petechial	Fibrin	Pulmonary

				agriculture labourer			House premises	12pm		activities							Blisters Cellulitis		Edematous	Haemorrhage	thrombi glomerulus Focal acute tubular Necrosis Mild Interstitial inflammation	edema
73.	1288/18																					
74.	1315/18	46	F	Agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Rainy	Household Activities	Lower Limb		13 Vials	6 hours	6 days	Present	Ulceration Cellulitis Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Sepsis, increased CPK Increased LDH
75.	1338/18	40	F	Agriculture labourer	Uneducated	Krait	Indoor	6pm to 12am	Rainy	Household Activities	Lower Limb				8 days	Absent	Cellulitis Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	Metabolic Encephalopathy, Sepsis.
76.	1380/18	53	M	agriculture labourer	Uneducated	Krait	Others	6 am to 12pm,	Winter	Agriculture Work	Lower Limb				8 days	Absent	Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
77.	1409/18	25	F	agriculture labourer	Primary School	Unidentified	Agriculture Field	12pm to 6pm	Winter	Agriculture Work	Lower Limb				6 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial Inflammation	
78.	1457/18	49	F	agriculture labourer	Uneducated	Viper	Agriculture Field	6pm to 12pm	Winter	Agriculture Work	Upper Limb				1 day	Present	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
79.	1499/18	23	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				10 days	Absent	Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	
80.	1518/18	25	M	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6am to 12pm	Winter	Agriculture Work	Lower Limb				1 days	Absent	Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Severe Interstitial inflammation	Intra cerebral haemorrhage
81.	1520/18	72	F	agriculture labourer	Uneducated	Unidentified	Houses Premises	12pm to 6pm	Winter	Others	Lower Limb				19 days	Absent	--		--	--	--	
82.	1549/18	38	M	agriculture	Uneducated	Unidentified	Indoor	6am	Winter	Sleeping	Upper				5 Hours	Present	Edema		--	--	--	

				labourer				to 12pm			Limb										
83.	1609/18	55	M	Non agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Winter	Others	Upper Limb				14 Hours	Present	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
84.	1648/18	66	M	Non agriculture labourer	Uneducated	Unidentified	Others	6pm to 12am	Winter	Others	Lower Limb				7 days	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage		
85.	1691/18																					
86.	1698/18																					
87.	1701/18	50	M	agriculture labourer	Uneducated	Unidentified	Indoor	6pm to 12am	Winter	Sleeping	Lower Limb				6 days	Present	Congestion Edema Gangrene		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis Congested Capillary loops	
88.	1724/18	16	F	Student	Higher Secondary	Cobra	Indoor	12am to 6am	Winter	Sleeping	Upper Limb		12 vials	6 hours	4 days	Absent	--		--	--	Nil	
89.	1758/18	51	M	agriculture labourer	Uneducated	Krait	Indoor	6pm to` 12am	Winter	House hold Work	Lower Limb				3 days	Present	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	
90.	1806/18	70	F	Non agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Winter	Others	Lower Limb				18 days	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	
91.	1851/18	62	M	Non agriculture labourer	Uneducated	Unidentified	Others	6am to 12pm	Winter	Others	Lower Limb				1 day	Present	Congestion Edema				Acute (diffuse) Tubular Necrosis	
92.	1888/18																					
93.	1891/18																					
94.	1905/18	35	F	agriculture labourer	Uneducated	Unidentified	Agriculture Field	6pm to 12am	Winter	Agriculture work	Upper Limb				1 day							
95.	1919/18	65	F	agriculture labourer	Uneducated	Unidentified	Indoor	12am to 6am	Winter	Sleeping	Lower Limb		12 vials	4 hours	8 hours	Absent	Congestion Edema		Congested Edematous	Petechial Haemorrhage	Acute (diffuse) Tubular Necrosis	
96.	1930/18	34	M	Non agriculture labourer	Uneducated	Krait	Indoor	12am to 6am	Winter	Sleeping	Gluteal region				3 days	Present						
97.	1948/18	5	M	Student	Primary School	Cobra	Indoor	6pm to 12am	Winter	Others	Upper Limb				1 day	Present						
98.	1960/18	46	F	agriculture labourer	Uneducated	Unidentified	Others	12pm to 6pm	Winter	Others	Lower Limb				7 hours	Present						
99.	1979/18	32	M	agriculture	Uneducated	Unidentified	Others	12pm	Winter	Others	Lower				3 days	Absent	Congestion		Congested	Petechial	Acute	

				labourer				to 6pm			Limb						Edema		Edematous	Haemorrhage	(diffuse) Tubular Necrosis	
100.	1998/18	55	M	agriculture labourer	Uneducated	Unidentified	Others	12am to 6am	Winter	Others	Lower Limb				5 days	Absent	Congestion Edema					
101.	2010/18	74	F	Agriculture labourer	Uneducated	Unidentified	Agriculture Field	12.PM	Winter	Work	Lower Limb	-			1 Day	Absent	-		Congested	Petechial haemorhage	Acute (focal) Tubular Necrosis	Subarachnoid Haemorrhage Intraventricular haemorrhage
102.	2025/18	55	M	Non agriculture labourer	Uneducated	Unidentified	Outdoor House premises	7pm	Winter	others	Lower Limb	-			10 Days	Present	Edema, blisters		Congested	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
103.	2059/18	15	M	Student	Higher secondary	Viper	Others	6pm-12am	Winter	Others	Lower Limb	-	35 Vials	7 hours	6 Days	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Disseminated Intra vascular Coagulation, Pulmonary Edema
104.	2073/18	55	F	Non agriculture labourer	Uneducated	Viper	Others	6pm-12am	Winter	Others	Lower Limb	-			3 Days`	Present	Edema, Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	
105.	2081/18	30	F	Others	Uneducated	Viper	Others	6am-12pm	Winter	Others	Lower Limb	-			3 hours	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Acute (focal) Tubular Necrosis	Pulmonary edema
106.	2095/18	58	M	Non agriculture labourer	Uneducated	Viper	Outdoor House premises	6am-12pm	Winter	Household activities	abdomen	-			9 days	Present	Edema, Blisters Cellulitis		Congested Edematous	Petechial Haemorrhage	Fibrin thrombi glomerulus Focal acute tubular Necrosis Mild Interstitial inflammation	Pulmonary edema
107.	2101/18																					